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CARCINOMA OF THE BREAST AT THE UNIVERSITY OF OKLAHOMA, 1948–1952*

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The purpose of this paper is to report the results of management of a series of patients with carcinoma of the breast treated at the University of Oklahoma Medical Center during the years 1948 through 1952 and to form a basis for comparison to later studies if desired. No attempt is made to review the literature concerning this subject since numerous excellent papers are available.[3-6] The dates 1948 and 1952 have been selected so that a minimum 5-year follow-up study could be made.

During this period 142 patients with carcinoma of the breast were seen on the Inpatient Service of the University Hospital at the University of Oklahoma. A study of the records of these patients led the author to believe that the Halsted type of radical mastectomy was not consistently employed until 1948. An analysis of the management of the patients with this disease has not been previously reported from the University of Oklahoma. Only 2 of the patients were males. Of the entire series, 19 were private patients, and these were treated by surgeons active on the teaching staff. With the exception of the private patients, all of the living patients were personally examined and interviewed by the author. Follow-up of the private patients was accomplished by examining the office records of their surgeons or the patient when possible.

The patients in this study came from all sections of the State of Oklahoma, although predominately from the Southeastern quadrant. Of the entire series 108 were Caucasian, 30 were Negro and 2 were Indian. In the state with the largest Indian population, except Arizona, this might be considered unusual but can probably be explained by the fact that the majority of Indian medically indigent patients are seen in the United States Public Health Service Hospitals in the State. During the period of study, almost all patients treated surgically were operated upon by second, third and fourth year residents in general surgery under supervision of the attending staff.

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CLINICAL FINDINGS

All of the patients were seen initially by practicing physicians in the State of Oklahoma who, after the approval of the local county commissioner, referred them to University Hospital. The duration of symptoms varied widely and seemed to have little significance in 5-year survival rate. The left breast was involved in 73 cases and the right breast in 61 cases. Both breasts were involved in 6 cases (fig. 1). In both breasts the upper lateral quadrants were the most frequent sites——36 in the left breast and 29 in the right breast. In 58 of the 101 patients who were treated by radical mastectomy, the tumor was greater than 3.0 cm. in diameter and in 43 patients it was 3.0 cm. in diameter or smaller. Although the objective findings were recorded, evaluation of operability from a study of the charts proved to be most difficult. It did seem, however, that a significant number of the patients had advanced disease. Of the 101 patients who underwent radical mastectomy 65 had axillary node metastasis, proved microscopically.

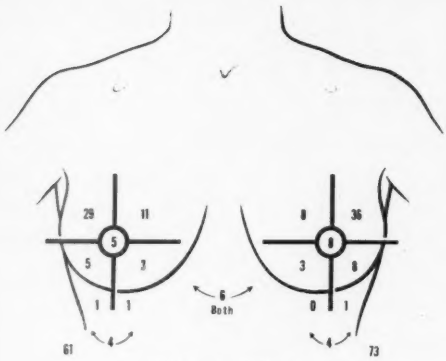

FIG. 1. Location

The patients ranged in age from the early third decade to the late ninth decade. The majority of the patients (80 per cent) were between 40 and 70 years old (fig. 2).

METHODS OF TREATMENT

Most of the patients were treated initially at the University Hospital; 17 of the patients had an incisional or excisional biopsy before referral, and 3 had prior simple mastectomies. These 3 patients had re-excision and radical axillary dissection after arrival at the University Hospital. The Halsted type of radical mastectomy was used in 101 of the cases. Halsted type of radical mastectomy is defined as radical en bloc dissection of the breast, pectoralis muscles, and the axillary nodes with axillary vein stripping. Simple mastectomy was performed on 9 patients. Radiation therapy alone was used in 21 cases, and 6 patients received no treatment.

Preoperative radiation therapy was not given in any case. Only 2 of the patients who had simple mastectomy received postoperative irradiation. In the group treated by radical mastectomy, 44 received postoperative radiation therapy. Of the patients who received radiation therapy after radical mastectomy 86 per cent had axillary metastasis. The x-ray given varied widely in timing and in dosage.

RESULTS

The results of treatment are known in all of the female patients in this series except 8, who were lost to follow-up. Of the 2 male patients in

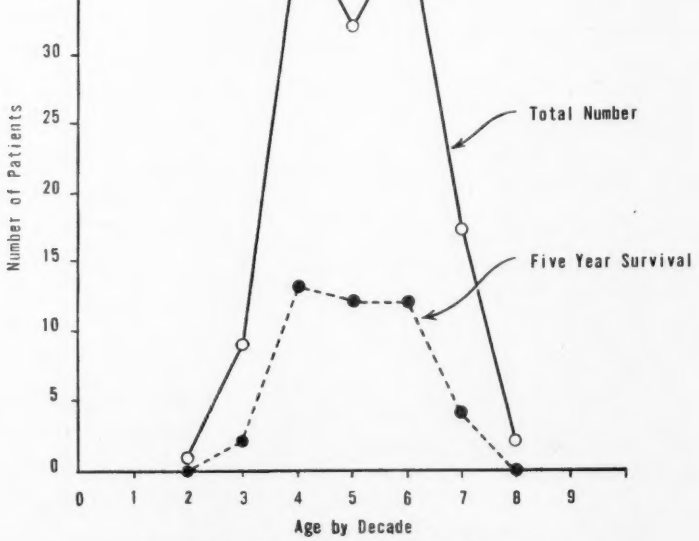

FIG. 2. Age distribution and survival by age

the series, one is living without evidence of residual carcinoma and the other is lost to follow-up. There was no operative or hospital mortality.

Of the entire 140 women, 35 are now living and 97 are dead; 8 are lost to follow-up. All of the living patients have been followed at least 5 years and all are free of residual carcinoma except one who developed a mass beneath her medial flap 10 years after radical mastectomy. This has been excised, and she has received x-ray therapy. In the group of 97 patients known to be dead, metastatic disease appeared to be the most common cause of death. However, most of these patients died in their home counties, and autopsy was rarely performed. Most of the patients died within 3 years after the date of their diagnosis. The 6 patients who received no treatment all died soon after their diagnosis. The average duration of life in these 6 patients was 9 months. One of these patients, because of religious beliefs, refused even biopsy, but obviously had far advanced carcinoma of the breast with distant metastasis. In the group of 21 patients who received only radiation therapy, all but one had far advanced disease and were referred to the radiotherapist by the surgeon. In no case was the x-ray dosage in the range now considered adequate. It is interesting that in the group of 9 patients treated by simple mastectomy only 2 received postoperative radiation therapy. Careful review of these charts revealed that in most of the cases the simple mastectomy was performed solely to relieve the patient of an ulcerating, foul, bulky tumor mass. The 2 patients who did receive postoperative radiation are still living and are without evidence of residual carcinoma. Both of these patients were private patients, and both had very small primary tumors present for less than 3 months (table 1).

The results of treatment by radical mastectomy are shown in table 2. Of these patients 42 lived 5 years, and 62 are now known to be dead; 65 of these patients had proved axillary metastasis and 36 were negative microscopically. Of the group with axillary metastasis, 27 received no postoperative irradiation, and the 5-year survival rate was 37 per cent, whereas it was only 23.7 per cent in the group of 38 patients who received postoperative radiation; 36 of the patients had negative nodes in the axilla, and the 5-year survival rate was 66.7 per cent irrespective of whether or not they received x-ray therapy.

TABLE 1

Survival after other methods

Management	Total	Lived 5 yr.	Per Cent	Dead	Lost	Per Cent Dead or Lost
Simple mastectomy	7	0	0.0	7	0	100.0
Simple mastectomy plus x-ray	2	2	100.0	0	0	0.0
Axillary dissection with positive nodes	1	0	0.0	1	0	100.0
Axillary dissection with negative nodes	2	1	50.0	1	0	50.0
X-ray only	21	1	4.8	20	0	95.2
No treatment	6	0	0.0	6	0	100.0

TABLE 2

Survival after radical mastectomy

Management	Total	Lived 5 yr.	Per Cent	Dead	Lost	Per Cent Dead or Lost
Radical mastectectomy	101	42	41.6	62	8	69.3
Positive nodes without x-ray	27	10	37.0	19	2	77.8
Positive nodes with x-ray	38	9	23.7	31	4	92.1
Negative nodes without x-ray	30	20	66.7	10	2	40.0
Negative nodes with x-ray	6	4	66.7	2	0	33.3

Because 17 of the patients were originally diagnosed by biopsy elsewhere and not by frozen section, it was considered pertinent to determine the 5-year survival rate according to time of definitive radical surgery; 80 patients were treated immediately after frozen section study of biopsy material. Of these patients 33 lived 5 years; 6 of the 17 patients biopsied elsewhere lived 5 years. Only 4 of the patients biopsied at the University Hospital had delayed radical mastectomy and 2 of these survived 5 years (table 3).

The 5-year survival rate was 55.8 per cent in the group of 43 patients who had a tumor mass 3.0 cm. or smaller, and 31.0 per cent in the 58 patients who had a tumor greater than 3 cm. There was no significant difference in survival when correlated with the duration of symptoms in the group who had radical mastectomy (table 4). The duration of symptoms before treatment ranged from 7 days to 10 years. The average duration of symptoms was 4 months.

COMPLICATIONS OF RADICAL MASTECTOMY

In most of the published literature little is said concerning the complications associated with radical mastectomy. Five types of complications, which occurred in this series of patients, were evaluated. Results are given in table 5. Wound infection occurred in 16 of the patients. Slough of wound edges or flaps was reported in 39 cases. The severity of the slough varied from a few millimeters to massive loss of wound flaps. Hematoma was mentioned in 13 cases. Edema of the arm after surgery was only reported 5 times, although in most cases no values were given for pre- and postoperative arm circumference. In the patients now living in this series, no significant difference was found by personal measurement. Pulmonary complications were rare and were reported only 3 times. Many patients had significant temperature elevations on the second and third postoperative days.

TABLE 3

Relationship of biopsy to time of radical mastectomy

Method	Total	Lived 5 yr.	Per Cent
Frozen section and immediate surgery	80	33	41.3
Biopsy elsewhere and delayed surgery	17	6	35.3
Biopsy at university and delayed surgery	4	2	50.0

TABLE 4

Relationship of size of primary and duration of symptoms to survival after radical mastectomy

Size and Duration	No.	Lived 5 yr.	Per Cent
Primary 3.0 cm. or smaller	43	24	55.8
Primary greater than 3.0 cm.	58	18	31.0
Symptoms 6 months or less	53	22	41.5
Symptoms more than 6 months	48	20	41.8

TABLE 5

Morbidity and mortality of radical mastectomy

Complication	Number	Per Cent
Infection	16	15.8
Slough	39	38.6
Hematoma	13	12.9
Pulmonary	3	3.0
Edema of arm	5	5.0
Mortality	0	0.0

DISCUSSION

The over-all survival for this entire series of 140 female patients was 33.6 per cent, which at first glance appears rather low but compares favorably with a much larger series reported by Hickey at the State University of Iowa.[2] This rate may possibly be due to the fact that the patients appeared rather late or with large tumors when first seen in the University Hospital. Also, it is obvious from a review of the figures that more than 19 per cent received only palliative radiation therapy or no treatment at all. During the period of study radical mastectomy was employed as the treatment of choice without rigid adherence to the criteria of operability, as suggested by others.[1] No attempt was made in this study actually to determine the operability from a study of the charts alone. Such an attempt is often difficult, and it is not believed that any conclusions could be drawn. It was of interest that many of the charts contained statements to the effect that the patient was inoperable by Haagensen's criteria, but radical mastectomy was chosen as the primary treatment.

The 5-year survival rate is paralleled closely by the age distribution of the patients (fig. 2). The series is too small to draw any conclusions, but it is suggested that carcinoma in the young woman is a more malignant disease than the carcinoma of the postmenopausal woman. One might also expect a higher death rate in the patient in the seventh and eighth decades because of the multiplicity of other diseases seen commonly in those age groups.

Of the patients with axillary metastasis who did not receive postoperative radiation 35 per cent survived 5 years, whereas only 23.7 per cent survived 5 years who received postoperative radiation. This may be explained by the fact that radiation therapy was more often employed postoperatively for the more advanced disease state. Five-year survival was the same in the patients

with negative nodes whether or not postoperative radiation was given.

Complications after radical mastectomy were numerous but none was fatal. The most common complication was slough of wound edges and/or flaps. Most of the patients who developed slough had more than one complication. No relationship was found between postoperative edema and slough or infection. The patients still living did not feel subjectively that they were disabled by having had radical mastectomy.

SUMMARY

1. A study of 140 female patients with carcinoma of the breast seen on the Inpatient Service at the University Hospital of the University of Oklahoma from 1948 to 1952 is given.

2. Except for 8 who were lost before 5 years, all patients were followed for a minimum of 5 years. All living patients were examined and interviewed by the author except for most of the private patients.

3. A Halsted type of radical mastectomy was performed in 101 of the patients. Simple mastectomy was performed 9 times. Twenty-one of the patients received palliative and x-ray therapy, and 6 patients received no treatment. Three patients had radical axillary dissections following simple mastectomy elsewhere.

4. Ninety-seven of the patients are known to be dead, and 8 are lost to follow-up. Thirty-five are still living.

5. The 5-year survival rate of the entire series of 140 patients was 33.6 per cent. The 5-year survival rate of the 101 patients treated by radical mastectomy was 41.6 per cent. There was a 5-year survival rate of 22.2 per cent in the group of 9 patients treated by simple mastectomy.

6. In the group managed by radical mastectomy 64 per cent had axillary metastasis and 36 per cent did not. The 5-year survival rate was almost the reverse of these percentages, being 29.2 per cent in the group with positive nodes and 66.7 per cent in the group with negative nodes.

7. The most common complication was slough of wound edges and/or flaps. Arm edema and functional disability were rare.

8. One patient who received radiation treatment only lived 5 years.

9. The need for early diagnosis when the tumor mass is small and the axillary nodes are free of metastasis is obvious.

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MANAGEMENT OF EXOPHTHALMOS*

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Progessive exophthalmos is the most distressing complication of thyrotoxicosis for both the physician and the patient. This condition usually occurs after definitive therapy for hyperthyroidism and is characterized by increasing scleral injection, conjunctival and retro-orbital edema, proptosis, ophthalmoplegia, diplopia, and it may lead to diminishing visual acuity, corneal ulceration and infection. It is possible, however, for this syndrome to reach full-blown proportions in patients who have not developed any other evidence of hyperthyroidism.[2, 3, 8-10] The responsibility of the physician is to arrest or reverse the process before loss of vision occurs. This report is concerned with our experiences in dealing with this truly gruesome complication at the Albany Medical Center.

Although the first studies of the etiology of this condition were those of Nauman[11] in 1853, there is still no unequivocal agreement as to the cause of malignant exophthalmos. Poncet[12] appears to have been the first, in 1910, to suggest that the pituitary was implicated. More recently, Dobyns[13, 14] showed that an exophthalmic-producing substance (EPS) in the anterior pituitary was different from the thyrotropic stimulating hormone (TSH).[15] The interrelationship among androgens, estrogens, cortisone and adrenocorticotropic hormone (ACTH) as etiologic possibilities have all been carefully studied, but final conclusions are still not available.[16-18]

Even the nomenclature of this problem is far from uniform. The terms, thyrotoxic and thyrotropic exophthalmos, are used to distinguish between the mild, almost always reversible eye signs, and the progressive permanent changes which constitute the severe form of this complication. Werner[8] refers to these differences as mild, non-infiltrative ophthalmopathy and severe infiltrative ophthalmopathy. Malignant exophthalmos[3, 6] or pituitarigenic oculopathy[19] are other terms used to describe the severe manifestations.

No matter which term you prefer, the pathologic changes are much better understood and almost unanimously agreed upon.[20-29] There is an absolute increase in the orbital content which pushes the globe forward. This is mainly an increase in fat and water which involves the extraocular muscles, especially the levator palpebrae.[21] There is edema with destruction and loss of normal architecture in these muscles with round cell infiltration.[30] Edema of the orbital tissues follows deposition of a mucopolysaccharide, provably hyaluronic acid.[31-34] These changes can result in fibrosis which may be evident within 6 months of the onset of proptosis.

As previously noted the ocular changes can appear in one eye or both before there is any evidence of disease in the thyroid gland. They can appear while the patient is hyperthyroid but most often they are associated with the postoperative or post-therapy hypothyroid state.[18, 34, 35] There are many who feel that the rapidity with which a hyperthyroid patient is returned to a euthyroid or mildly hypothyroid state influences the onset or infiltrative ophthalmopathy. Characteristically, there is limitation of upward and then outward gaze. Proptosis becomes evident; and as the problem progresses, marked to complete limitation of movement of the globe may result. Edema of the conjunctivae, injection of the sclerae, lacrimation, a feeling of retro-orbital pressure and of "sand" in the eyes are common complaints. When the lids can no longer close over the eye, corneal drying, ulceration and infection may follow and lead to loss of the eye.[4] Visual acuity may diminish as intra-orbital pressure rises causing compression of the optic nerve. The physician-in-charge is obligated to institute measures to prevent loss of sight and loss of the eye. The changes in vision may advance with great rapidity leaving no time for prolonged observation before carrying out definitive treatment.

Tarsorrhaphy, supraorbital or lateral orbital decompression have been standard procedures to contain the globe and protect its cornea, or to provide an increase in the space for the orbital

* From the Radioisotope Laboratory of the Albany Medical Center.

contents.[1, 2, 5, 37] Pituitary irradiation by external roentgen rays, similar radiation to the retro-orbital contents, use of androgens, estrogens, cortisone, ACTH and thyroid hormone have all been tried as nonsurgical measures to overcome the problem of exophthalmos.[2, 7, 36] McCullagh and associates have indicated some success with ACTH and cortisone in a small series of cases. For those patients in their reproductive years this medical treatment or supraorbital decompression is preferred to pituitary ablation. Destruction of the anterior pituitary by surgery or yttrium[90] implantation, cauterization or division of the pituitary stalk are more recent surgical procedures employed to relieve this distressing problem.[1, 5, 6, 17, 37]

There seems to be little doubt that anterior pituitary destruction offers the most promising results and the best opportunity for arresting the progress of malignant exophthalmos. The return to near normal of all changes in the eyes following pituitary surgery is dramatically rapid and proportionately gratifying to patient and doctor alike.

Case A. M. M. G., a 66-year-old woman, was first seen on August 14, 1958, with complaints of puffy eyes and blurred vision of 2 weeks' duration. Vertigo, weakness, nervousness and sweating had been present 4 weeks. Palpitation, exertional and nocturnal dyspnea with ankle edema had been noted for 1 year. The patient was normotensive, had ophthalmoplegia, severe exophthalmos, cardiomegaly, auricular fibrillation and cardiac insufficiency. Routine laboratory studies normal; electrocardiogram abnormal with auricular fibrillation, old anterior myocardial infarct and left ventricular hypertrophy. Cholesterol was 366, esters 76 per cent; protein-bound iodine 5.1 mcg. per cent; radioactive iodine uptake 34 per cent at 24 hours; skull films showed no abnormality. Satisfactory total hypophysectomy was done on August 22, 1958, because of further loss of vision. The postoperative course was uneventful, and the patient went home on September 3, 1958, with recovery of vision, marked remission in scleral injection and conjunctival edema and diplopia, but with residual lateral rectus palsy.

Case B. M. E. D., a 50-year-old woman, was first seen on March 30, 1957, when a diagnosis of toxic nodular goiter was made and preoperative treatment started. Thyroidectomy was carried out on May 20, 1957, and the pathology report was: "Multiple adenomatous colloid goiter with foci of cystic degeneration, hemorrhage, fibrosis and foci of active hyperplasia." On June 30, 1957, diplopia was noted and thyroid extract started. On August 12, 1957, left thoracotomy for hiatus hernia was accomplished without incident. In March 1958 the exophthalmos was progressing in spite of suppressive therapy, and the diplopia was more pronounced. On March 20, 1958, hypophysectomy was done with an uneventful recovery and rapid return of the eyes to normal.

When we failed to bring about a remission in the progress of this disorder using suppressive, exogenous thyroid hormone therapy, the suspicion arose that any active normal thyroid gland might be contributing to our lack of success. This feeling was based upon observations that revealed the difficulty of producing experimental exophthalmos in animals possessing an intact thyroid gland as compared with those which had been thyroidectomized.[24, 34] Data indicating that the thyroid gland inactivates endogenous thyrotropin and that functioning thyroid tissue may alter the response of other body tissues to the thyroid hormones[38, 39] enhanced our belief that functioning thyroid tissue was a factor in our inability to suppress production of EPS with exogenous thyroid hormone.

We have long felt that the histologic variations in surgically excised thyroid tissue helped to explain why some patients responded well to operation or radioactive iodine and why others became markedly hypothyroid. If the thyroid gland is able to inactivate TSH and is not influenced by EPS, this may help to account for those rare patients with marked exophthalmos and little or no hyperthyroidism.[40] The theoretical possibility of these interrelationships is emphasized clinically when exophthalmos progresses while the patient receives near toxic doses of exogenous thyroid hormone as documented by Brain[3] and Bruun[4] and others.

It seemed reasonable to us on the basis of the above clinical and experimental data that the management of such patients could be more easily accomplished if all normal thyroid tissue was ablated with I^{131}. Those patients selected for this approach were given 30 mc. of I^{131} by mouth after suitable withdrawal of other therapeutic measures. A total of 8 patients have now been treated in this fashion in an attempt to control their disease, and this group has been followed for over 6 months. No untoward effects attributable to the I^{131} therapy have been observed. Reinstitution of suppression therapy with triiodothyronine has been carried out 1 to 2 weeks

TABLE 1

Improvement by symptoms after thyroid gland ablation with I^{131}

	I C. S.		II H.M.		III N.H.		IV A.M.C.		V H.H.		VI D.H.		VII V.T.		VIII E.S.	
	Pre	Post[a]	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Injection	4+	0	4+	1+	4+	1+	4+	1+	4+	2+	4+	1+	4+	1+	4+	1+
Edema	3+	0	4+	0	2+	0	4+	0	4+	2+	4+	0	4+	0	4+	1+
Lacrimation	3+	1+	4+	1+	4+	0	4+	1+	4+	2+	4+	1+	4+	0	4+	2+
Diplopia	0	0	3+	±	2+	0	2+	0	0	0	4+	1+	1+	0	0	0
Ophthalmoplegia	4+	0	3+	1+	3+	1+	4+	1+	4+	3+	4+	1+	3+	0	0	0
Visual acuity	↓	N[b]	↓	IMP[c]	↓	N	↓	IMP	0	0	↓↓	N	N	N	↓	N
Corneal ulceration	0	0	0	0	0	0	1+	0	0	0	2+	0	0	0	0	0

[a] Pre = preoperative; post = postoperative.
[b] N = normal
[c] IMP = improved

after the I^{131} administration. Triiodothyronine has been rapidly raised to toxic levels, then reduced and continued at a less than toxic dose, usually 150 to 250 mcg. per day.

Patient-selection has been restricted to those whose eye signs and proptosis were progressing in spite of adequate exogenous thyroid hormone therapy. Selection of the time to advise ablation depends upon the rapidity with which scleral injection, conjunctival edema, ophthalmopathies and proptosis progress. Although we are opposed to irradiation of the thyroid by I^{131} in young patients because of carcinogenic possibilities, we feel that completely destroying the thyroid as a means of saving vision has more advantages than hypophysectomy in this younger age group.

Although there has been marked individual variation in response to this program, all have exhibited satisfactory cessation of progress of their exophthalmos. As noted in table 1, virtually all patients have shown improvement in every major symptom. Scleral injection has shown from 50 to 100 per cent improvement. Conjunctival edema has similarly shown the same degree of regression. Lacrimation has not benefited to the same degree, but diplopia has been almost completely abolished. Muscle weakness or paresis has always improved but not always as rapidly nor as completely during the first 6 months of postablation observation. Table 2 indicates that there usually has been a slight regression in the proptosis. The question marks before the reading of DH are required because of a lateral orbital decompression with resultant loss of the orbital ridge and no solid base from which to measure. The summaries of these patients are presented below.

TABLE 2

Changes in measured proptosis after I^{131} ablation of the thyroid using Hertel exophthalometer

Patient	Age	Sex	Before Treatment		At Last Visit	
			OD*	OS*	OD	OS
C. S.	51	F	23	23	24	24
H. M.	54	F	29	29	28	28
N. H.	48	M	27	27	24	24
A. M. C.	44	F	26	25	24	24
H. N.	54	F	27	27	24	24
D. H.	19	F	?28	?28	?26	?26
V. T.	43	F	25	26	25	25
E. S.	50	F	25	25	22	23

* OD, right eye; OS, left eye.

Case 1. C. S., a 51-year-old woman, had a preoperative diagnosis of toxic nodular goiter because of greatly enlarged right thyroid lobe and normal sized left. Thyroidectomy was performed on March 10, 1958. Pathology: "Multiple areas of hyperplasia, involution, lymphocytic infiltration and lymph follicle formation." Preoperative proptosis measured on the right eye (OD) 19, on the left eye (OS), 19; increased to OD 21, OS 21 postoperatively. She was treated first with thyroid extract, then triiodothyronine, without remission in eye signs. Proptosis increased to OD 25, OS 25. Ablation of the thyroid was accomplished with the use of 30 mc. of I^{131} on February 12, 1959.

There was gradual remission of scleral injection and of the edema. Minimal restriction of upward-outward gaze remains. Proptosis measures OS 24, OS, 23.

Case 2. H. M., 55-year-old woman, first seen on August 21, 1957, had a history of hyperthyroidism for 10 months. She was treated with Tapazole for toxic diffuse goiter. Initial proptosis measured OD 25, OS 25. Exophthalmos increased to OD 27, OS 26. The patient was treated with 10.26 mc. of I^{131} on October 16, 1957. Treatment repeated on December 3, 1957, with 9.09 mc. of I^{131}. The right thyroid lobe continued to enlarge causing tracheal deviation, and the hyperthyroidism continued. Thyroidectomy was performed April 7, 1958. Pathology: "Diffuse hyperplasia of thyroid gland."

Proptosis measured OD 27, OS 27 postoperatively. Triiodothyronine was given for 4 months, but the eyes became more protruberant with exophthalmometer readings of OD 29, OS 29. Thyroid ablation was accomplished with 30 mc. of I^{131} on September 23, 1959. Four months post-ablation there is decreased lacrimation with only a trace of scleral injection and conjunctival edema. Exophthalometer readings on March 16,1960, were OD 26, OS 25.

Case 3. N. H., a 49-year-old man, was first seen January 20, 1956, when the diagnosis of toxic diffuse goiter was made. Early eye signs were present, and proptosis of OD 23, OS 23 noted. Thyroidectomy was performed on February 21, 1956. Exogenous thyroid hormone was started in March but exophthalmos progressed. In October 1956 proptosis measured OD 25, OS 25. Partial tarrsoraphy was done in March 1957. The eyes progressed to OD 27, OS 27 in May 1958. The thyroid gland was ablated with 30 mc. of I^{131} on May 29, 1959 and triiodothyronine restarted.

After ablation a gradual remission of edema and injection occurred. The extraocular movements returned toward normal. Proptosis in November 1959 measured OD 24, OS 23 while receiving 200 mcg. of T_3 daily.

Case 4. A. M. C., a 44-year-old woman, was first seen on January 21, 1959, with typical symptoms and signs of toxic diffuse goiter. Eye signs were marked at first examination with proptosis of OD 24, OS 23. The patient received 3.8 mc. of I^{131} on January 27, 1959. Hyperthyroidism improved slowly over the following 3 months, but the eye signs progressed. Blurring of vision occurred when a I diopter papilledema was observed. On April 7, 1959, 30 mc. of I^{131} was given at which time the exophthalmos measured OD 25, OS 25. After ablation, the scleral injection, conjunctival edema and the external ocular movements all improved. Patient is receiving 200 mcg. of triiodothyronine, and the proptosis measured OD 24, OS 24 on March 4, 1960.

Case 5. H. H., a 54-year-old woman, was first seen on January 16, 1959, with a 1-year history consistent with a diagnosis of toxic diffuse goiter. Eye signs were noted for 6 months which were initially recorded as limitation of upward-outward gaze, scleral injection, conjunctival and periorbital edema and lacrimation. Exophthalmos measured OD 25, OS 25. The patient received 3.4 mc. of I^{131} on January 27, 1959, with return to euthyroid state but had no regression of the eye change during the ensuing 6 months.

Ablation of the thyroid gland with 30 mc. of I^{131} was carried out on July 7, 1959. Subjective improvement was noted in 1 month with the proptosis measuring OD 24, OS 24. Slow improvement continued over the next 6 months to a trace of scleral injection with no conjunctival edema and residual limitation of motion. Proptosis on March 9, 1960, measured OD 26, OS 26. Triiodothyronine was increased to 200 mcg. daily.

Case 6. D. H., a 16-year-old girl, was first seen on August 17, 1956, with symptoms of hyperthyroidism and eye changes of about 2 months' duration. Initial proptosis measured OD 21, OS 21. Thyroidectomy was done on November 24, 1956. Pathology: "Diffuse hyperplasia." Eye signs progressed and tarsorrhaphy was done on the right eye in December followed by bilateral supraorbital decompression in April 1957. Exogenous thyroid hormone had been given and was continued. Right lateral orbital decompression was done in September 1957. An ablating dose of I^{131} (30 mc.) was given May 15, 1958, followed by reinstitution of suppressive therapy using triiodothyronine. Diminution in lacrimation and scleral injection was noted within a month. For the past 10 months further improvement has occurred so there is no longer any injection, less edema and some regression in proptosis.

Case 7. V. T., a 40-year-old woman, was seen for the first time on April 9, 1956, with symptoms and findings of toxic nodular goiter of about 3 months duration. No eye signs noted initially. Thyroidectomy was done on May 14, 1956, and the pathologist reported, "Multiple adenomatous colloid goiter with diffuse hyperplasia. Only mild involution is present." Periorbital edema noted in June 1956 and thyroid hormone started. In September her proptosis measured OD 22, OS 22 and periorbital edema was marked. On February 20, 1957, she was considered euthyroid (I^{131}, 24-hr. uptake, 36 per cent) but with progressive exophthalmos. On June 12, 1957, she received 9.17 mc. of I^{131} when her proptosis measured OD 25, OS 25. Following this treatment there was improvement in the lacrimation and burning sen-

sations but no change in the ophthalmoplegia or proptosis. Because the next year brought about no improvement in the eye signs, did result in thyroid gland enlargement, a diagnosis of persistent hyperthyroidism was made and surgical reduction in thyroid gland size with I^{131} ablation planned. The second thyroidectomy was carried out on May 26, 1958, when the pathologist again reported diffuse hyperplasia. On July 23, 1958, she received 30 mc. of I^{131} at a time when her proptosis measured OD 27, OS 26. Following this therapy there has been objective diminution in scleral injection and conjunctival edema. She is now being maintained on exogenous thyroid hormone without evidence of injection, edema or ophthalmoplegia. Exophthalomometer measurements on March 16, 1960, were OD 24, OS 25.

Case 8. E. S., a 47-year-old woman, was first seen on April 25, 1956, with a 5-month history of toxic diffuse goiter. Thyroidectomy was done on June 1, 1956, the specimen of which the pathologist reported, "Diffuse hyperplasia of the thyroid gland. There is only moderate involution." The initial Hertel exophthalomometric reading was OD 20, OS 21. On June 28, 1956, upper lid edema and lachrimation were evident and Proloid 1 gr. per day prescribed. This was increased to thyroid extract, 90 mg. per day, and finally to 120 mg. per day. On December 3, 1958, her proptosis measured OD 24, OS 25 with scleral injection, conjunctival edema and photophobia. Triiodothyronine started and continued for 5 months without improvement in ocular signs. Because of this failure to improve, 30 mc. of I^{131} were given on January 1, 1959, when the proptosis measured OD 24, OS 23. During the ensuing 12 months all eye signs have disappeared under triiodothyronine suppressive therapy and, the last measurement of her proptosis in early 1960 was OD 22, OS 22.

The financial advantage of this ambulant step in the management of progressive exophthalmos is obvious. When successful in arresting the advance of eye changes and preserving visual acuity, the patient avoids the expense of hypophysectomy and its postoperative, life-long hormonal replacement therapy. No one should hesitate to advise pituitary surgery if vision is in any way embarrassed. We again emphasize the importance of very close observation when this condition is in its acute phase. Vision may be lost with great rapidity compelling either pituitary surgery or supraorbital decompression virtually as emergency procedures. We believe that ablation of the thyroid with I^{131} and exogenous thyroid hormone therapy can reduce the number of patients who require these more extensive surgical procedures.

SUMMARY

1. Hypophysectomy offers the patient with malignant exophthalmos an opportunity to preserve vision and undergo reversal of most of the findings associated with such severe proptosis.

2. As preliminary treatment before such radical surgery is advised, ablation of the thyroid gland by radioactive iodine and full therapeutic doses of exogenous thyroid hormone may enable many patients to avoid pituitary surgery.

3. For "teen-agers" and patients in the early productive years, orbital decompression is preferred to operative procedures on the hypophysis in the event that I^{131} ablation and exogenous thyroid hormone do not control the disease.

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RESECTION OF THE CAROTID ARTERIES FOR NEOPLASTIC INVASION WITH MAINTENANCE OF CIRCULATION*

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Surgical resection of the common or internal carotid arteries, or operative interruption of their blood flow, is followed by a significant morbidity and mortality. Moore and Baker[9] after reviewing 88 patients who had had ligation of the common or internal carotid arteries, reported an over-all incidence of cerebral complications in 45.4 per cent of the cases and a mortality of 30.6 per cent. Their figures have been reduced to 31.4 per cent and 11.4 per cent respectively since 1950. DeBakey and Crawford[3] found the rate of complication after ligation of the main carotid stream to be 25 to 70 per cent. In their report 4 of 7 patients, who had had temporary interruption of the circulation followed by restoration of arterial continuity, suffered transient or permanent neurologic signs.

Consideration of these complications may deter the surgeon from proceeding with resections involving the common or internal carotid arteries. If he leaves this vital vessel with tumor infiltrating it, he must rely on external or interstitial radiation therapy to repress the remaining neoplasm.[6] It is generally conceded, however, that external radiation does not cure metastatic neoplasm in the neck while it may produce deleterious effects on the adjacent tissue, such as delayed healing, infection and even fatal hemorrhage. These and other considerations emphasize the advantages of total excision of all neoplastic tissue when possible and within the limits of the patient's safety. The development and use of a simple, inexpensive, temporary arterial bypass has aided in the achievement of this goal and has permitted the resection of the common and internal carotid arteries, as well as the restoration of flow in the carotid arteries by a replacement graft.

MATERIAL AND METHODS

The bypass shown in figure 1 was constructed using standard intravenous plastic tubing. Its rubber ends were attached to Luer-lok adapters, so that an intermittent, pulsatile flow could be maintained. A side limb was inserted near the proximal end by means of a Gilson mixing adapter to permit a constant slow infusion of heparin under pressure, using a 30-cc. or 50-cc. syringe. The dilute mixture contained 30 mg. of heparin in 100 cc. of saline. It was unnecessary to give over 50 cc., and this prevented an effective systemic anticoagulation level. Standard intravenous needles were cut off leaving a 1-in. shaft. The bevel was short and the point blunted to prevent accidental penetration of the arterial wall. The needle sizes extended from 12 to 17 gauge and two of each size were maintained in this set. The variation in diameter of the internal carotid artery made this selection necessary, but the use of the larger needles would be preferable. This set has been made available on all radical dissections of the neck. Neoplastic invasion of the common or internal carotid artery may be found rather late in the dissection after exposure of the carotid bifurcation or the internal carotid artery, those portions which unfortunately are most commonly involved. When neoplastic infiltration was found, the dissection was completed about the periphery, leaving the tumor and specimen attached to the vessels. The infiltrated vessel and specimen were then excised *en bloc* (figs. 2 and 3). To excise and replace the artery, a stump of normal vessel, 1.5 to 2 cm. in length, above the tumor, was needed. The vessels were isolated and noncrushing ligature was passed about the proximal and distal vessels. The largest needle which could be inserted into the vessels was used. The syringe and bypass were filled with the heparin-saline solution and all air expelled. Mosquito clamps were then applied to the rubber tubing at the ends. The needles were inserted well into the proximal vessel and then into the distal vessel bypassing the involved segment. As this was done, blood flow continued through the artery about the needles, thereby maintaining cerebral circulation. Security against

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dislodgement was obtained by the noncrushing ligatures. At this point, the by-pass clamps were removed, and a pulsatile cerebral circulation occurred through the bypass. An assistant injected the dilute heparin solution slowly to prevent coagulation within the tubing.

Potts vascular clamps were applied to the artery about the neoplasm and within the bypass, and the diseased segment was excised. The graft was then inserted. In the 2 cases to be reported, a preclotted Edwards-Tapp graft of the proper diameter and length was used. Completion of the distal anastomosis first was advantageous, as the graft was elevated to facilitate placing of the posterior high distal suture line. Two 5-0 continuous arterial silk sutures were used for the anastomoses, one posteriorly and one anteriorly between end traction sutures. The proximal anastomosis was facilitated by rotating the graft about its longitudinal axis. The ligatures about the needles were then cut. The proximal clamp was removed first. The flow of blood against the distal clamp expels any residual air, thus preventing cerebral air embolus. The distal clamp was then removed and then the needles.

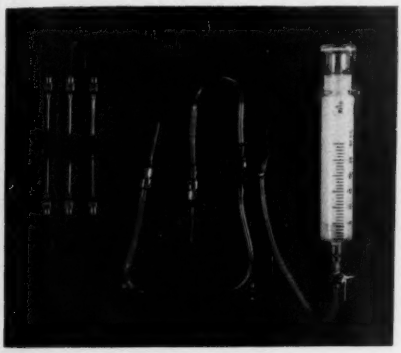

FIG. 1. Temporary arterial bypass shunt with a syringe to provide local anticoagulants to prevent blood coagulation.

In the 1st patient, we relied upon pressure dressings and an upright position to create a suitable nutrient bed for the graft. In the 2nd case, the posterior belly of the digastric muscle was separated from the hyoid bone and sutured about the graft as illustrated in figure 3. If this muscle had been inadequate, flaps would have been created from the sternohyoid, sternothyroid, or other adjacent muscles.

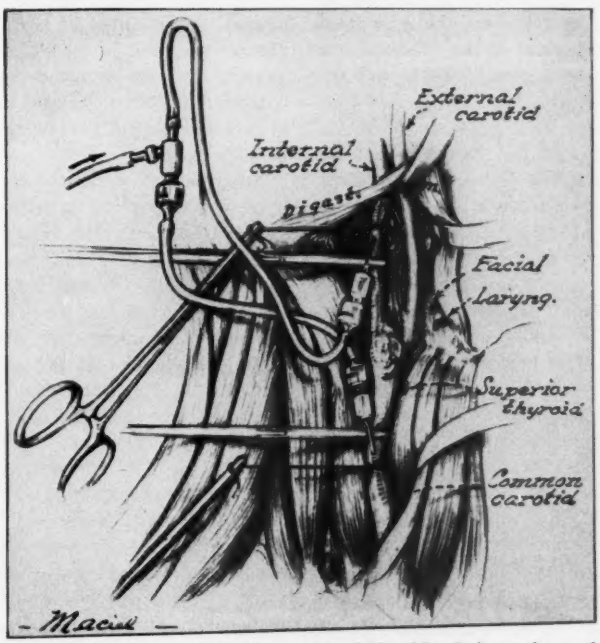

FIG. 2. The temporary arterial bypass is in place transmitting blood about the occluded arterial segment to be excised.

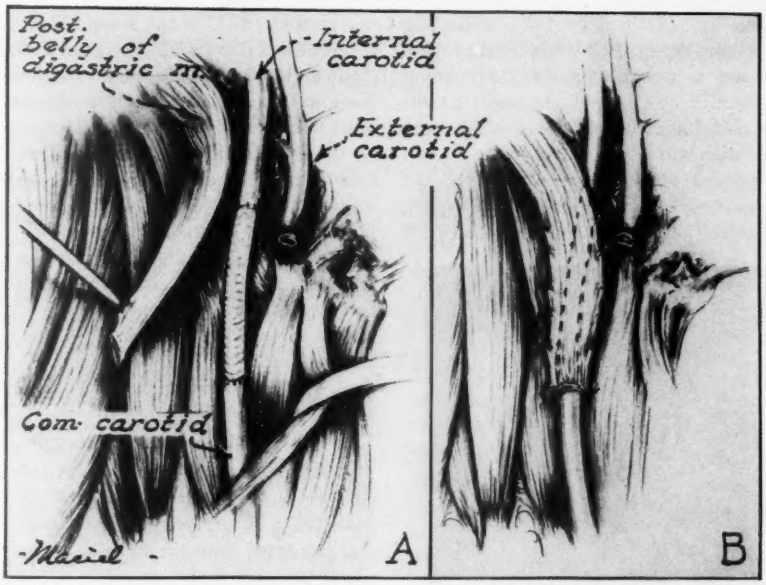

FIG. 3. *A*. The diseased arterial segment has been excised and replaced with a nylon prosthesis. The posterior belly of the digastric muscle has been separated from the hyoid bone. *B*. The posterior belly of the digastric muscle is sutured about the arterial prosthesis surrounding it in a muscle bed.

The variance in tolerance of the brain to occlusion of the main carotid flow and the subsequent sequela ultimately depends on the collateral flow provided by the contralateral vessels and the circle of Willis. Woodhall and associates[11] state that anomalies of the circle of Willis are seen in 50 per cent of dissections. Preoperative tests, such as the Matas' test to evaluate this collateral flow are inaccurate, especially when they suggest adequate cross circulation. Woodhall and coworkers[11, 12] measured directly the distal internal carotid pressure after temporary proximal ligation of the internal or common carotid arteries and found in 26 adults an average drop of 51 per cent in the systolic pressure, 31 per cent in pulse pressure, and 57 per cent in the integrated mean pressure. In cases where rupture of an aneurysm had made ligation of the carotid artery necessary, they concluded that pressure falls exceeding 50 per cent were frequently followed by neurologic impairment or death. Although these pressure determinations may reflect cerebral blood flow, they do not measure it. It is difficult to find actual measurements of cerebral blood flow. Kety,[5] using N_2O and dye injections, calculated the average cerebral flow to be 750 ml. per min., assuming a brain weight of 1400 g. Cooley[2] estimated the flow through one normal carotid artery at basal conditions to be 250 cc. per min. More accurate measurements await the refinement of newer techniques. Great variations in flow are suggested by the differences in the diameters of the carotid arteries and the blood pressures.

The blood flow transmitted by the arterial bypass described was tested in the laboratory. Mongrel dogs weighing from 28 to 56 lbs. were anesthetized with a 2.5 per cent solution of sodium Pentothal. A right low abdominal incision was made to mobilize the iliac arteries between the inguinal ligament and the aortic bifurcation. A 2nd longitudinal incision was used to expose the femoral artery throughout the right thigh. The artery was cannulated distally, and attached to a mercury manometer. After the pressure had been recorded, the proximal end of the femoral artery was divided and the blood collected to measure the flow. The plastic bypass using the 12-gauge needles was inserted proximally in the common iliac artery and distally in the femoral artery in the occluded area, proximal to the manometer. Further measurements of the pressure, by way of the bypass, showed that the normal pressure was maintained in every instance.

TABLE 1

Femoral blood pressure and flow through the bypass in five animals

Weight	Normal Pressures	Normal Femoral Blood Flow	Pressure Through Bypass	Blood Flow Through Bypass
lbs.	*mm. Hg.*	*cc. per min.*	*mm. Hg*	*cc. per min.*
36.5	120–140	560	110–120	176
48	130–135	504	130–135	162
28.5	120–125	450	120–130	180
34	130–140	510	130–140	190
38	120–130	500	120–130	180

The distal needle was then withdrawn, and the blood flowing through the plastic tube was collected and measured (table 1). The flow rate was variable but sufficient to maintain an adequate circulation.

CLINICAL CASES

Case 1

A 69-year-old white man was admitted to the Cincinnati Veterans Administration Hospital Medical Service on March 7, 1958, complaining of slight dysphagia and anorexia of several months duration. Recently he had also developed a chronic purulent cough. This patient had been previously confined to a chronic disease hospital with a diagnosis of chronic arthritis and chronic brain syndrome secondary to cerebral arteriosclerosis. He had a poor history.

Physical examination. Temperature 103.0°F., pulse 120, blood pressure 180/50. The patient was cachectic and coughed up copious amounts of a creamy yellow sputum. The left lower chest was dull to percussion and transmitted diminished breath sounds. There was a 3 by 2 cm. hard, firm cervical mass deep to the middle third of the left sternocleidomastoid muscle.

Laboratory data. White blood cell count 11,000 with 69 per cent neutrophils, 23 per cent lymphocytes, 6 per cent monocytes, 1 per cent basophils; hemoglobin 12.4 g., hematocrit 40 per cent; coagulation time 7 min.; urinalysis negative; prothrombin 78 per cent; total protein 6.35 gr. per cent, 2.7 g. albumin and 2.65 g. globulin; blood calcium 9.2 mg. per cent and phosphorus 3.85 mg. per cent.

Roentgen studies. Chest: Increased bronchovascular markings suggestive of bronchiectasis with left lower lobe pneumonia. Barium swallow suggested a mass in the region of the cricopharyngeal muscle.

As the pulmonary infection was brought under control with antibiotic treatment, a search was made for the primary site of the probable cervical metastatic tumor. Direct laryngoscopy revealed

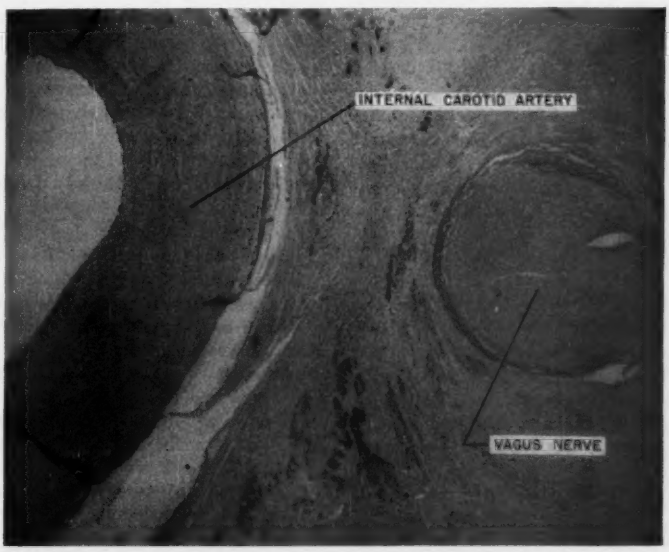

FIG. 4. This microscopic section shows the squamous cell carcinoma with its severe desmoplastic reaction completely surrounding the carotid artery and vagus nerve. The perineural lymphatic metastases are present.

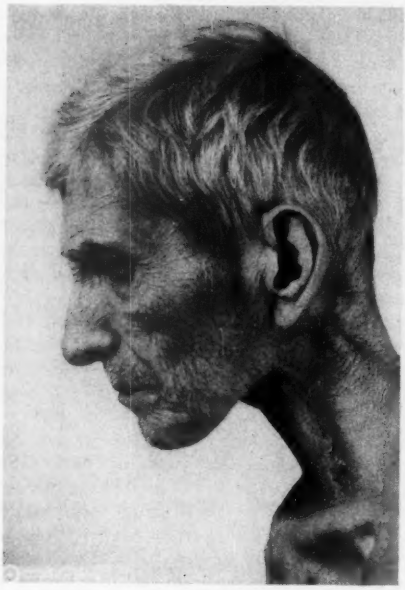

FIG. 5. The postoperative photograph demonstrates the lack of deformity in the position of the tracheostomy to the right of the esophagostomy to the left.

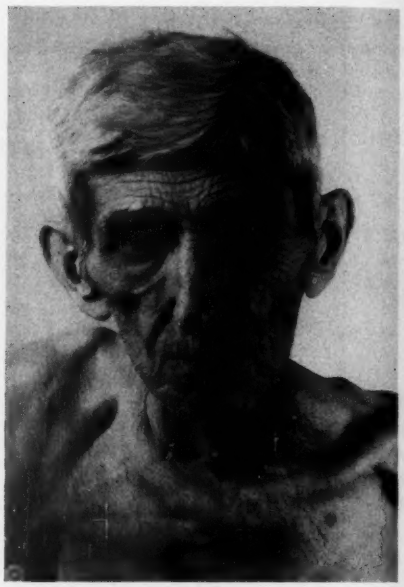

FIG. 6. This postoperative photograph demonstrates the lack of deformity in the position of the tracheostomy to the right of the esophagostomy to the left.

a fungating lesion in the left pyriform sinus with extensive invasion of the larynx, which caused his abnormal swallowing, the aspiration of pharyngeal contents and pulmonary infection. Biopsy revealed a squamous cell carcinoma. No distant metastases were found. On March 30, 1958, a wide-field total laryngectomy, left thyroid lobectomy and left radical neck dissection was undertaken. The lesion had extended well down into the esophagus. Considering the extensive nodal involvement, the extent and duration of the procedure, and the age of the patient, a cervical esophagostomy was created to the left of the tracheostomy. The posterior wall of the pharynx was approximated to the base of the tongue. The tumor had involved the internal carotid artery and vagus nerve (fig. 4) cephalad to the carotid bifurcation. These structures were excised using the arterial bypass and technique described above. The carotid artery was reconstructed with a crimped nylon prosthesis, using an end-to-end anastomosis. The shunt circulated the blood for 15 min. while the carotid vessel was excised and the prosthesis inserted. The entire specimen was resected in continuity.

Postoperatively the flaps healed *per primam*. The graft remained pulsatile until his death 7 months later, and at no time were neurologic complications seen (figs. 5 and 6). The excision of the larynx and closure of the pharynx greatly improved his pulmonary disease, and the tube feedings were easily given through the esophagostomy.

In September 1958 it became apparent that he had mediastinal paraesophageal metastases. He lost weight rapidly and expired on October 2, 1958. Autopsy confirmed the presence of mediastinal metastases. The graft was patent at autopsy.

Case 2

A 70-year-old man was admitted to the Cincinnati Veterans Hospital on June 3, 1958. In the spring of 1956, he had noticed a sore white "blister" on the floor of his mouth between the base of the tongue and the right mandible. In October 1956 a biopsy had revealed leukoplakia. He discontinued smoking and was treated with large doses of vitamin A. There was no improvement and recently it became more tender.

Physical examination. Temperature 98.6°F., pulse 80, blood pressure 124/80. There was a bluish white, slightly tender plaque with mimimal induration on the right margin of the tongue at the junction of the middle and anterior third. Inferiorly at the middle third of the base of the tongue was a 3 by 2 by 2 cm. tender nonfixed mass.

FIG. 7. This excised specimen shows the neoplastic infiltration at the bifurcation of the carotid artery.

It extended into the floor of the mouth and was ulcerated. No teeth were present. A firm tender node 2 by 1 by 1 cm. was felt in the right jugular chain opposite the hyoid bone. In addition a small umbilical hernia and a large left inguinal hernia were present. The clinical impression was squamous cell carcinoma of the base of the tongue with cervical lymph node metastases.

Laboratory data. Hemoglobin 15 g. per cent, hematocrit 48 per cent; white blood cell count 13,950 with 59 per cent neutrophils, 26 per cent lymphocytes, 11 per cent monocytes and 4 per cent eosinophils. Urinalysis revealed only 80 to 100 white blood cells. Blood urea nitrogen 17 mg. per cent. Total protein 6.6 g. per cent with 3.4 g. per cent albumin and 3.2 g. per cent globulin.

Roentgen studies. Chest plate revealed no abnormalities or visible metastases. Films of the mandible showed no evidence of neoplastic invasion.

A repeat biopsy of the mouth lesion on admission confirmed the clinical impression of squamous cell carcinoma. On June 19, 1958, the mouth lesion was implanted with radium needles. A total weight of 26 mg. was used for 118 hr., giving approximately 6000 gamma roentgens. The lesion responded very well; the ulceration healed and was covered by an epithelite. The node or mass in the neck continued to grow and was more painful. A right radical neck dissection was advised. This was performed on July 28, 1958. At operation the tumor was found to involve the hypoglossal nerve, and a segment of it was excised. Exposure of the carotid bifurcation and internal carotid artery during the dissection revealed residual tumor in this area. The bifurcation and a segment of the internal carotid artery with the contained neoplasm were therefore resected (fig. 7). A crimped nylon graft was inserted using the bypass and the described technique. The graft was covered with a muscular flap.

Postoperatively the patient showed no evidence of cerebral damage. He developed an infection under the skin flaps but this was brought under control. The wound healed, the graft has continued to pulsate, and no neurologic signs have been observed.

The patient developed a glossopharyngeal nerve tic which was triggered by swallowing. This disappeared spontaneously. This tic plus the disability caused by the hypoglossal nerve excision, caused malfunction of deglutition. A temporary gastrostomy improved his nutrition as his speech and swallowing have improved.

DISCUSSION

Various methods have been used to protect the central nervous system during temporary interruption of the carotid arterial blood flow. Lesions such as atheromata and thrombi, which have already obstructed the carotid flow, or lesions which stimulate collateral flow need less protection. Hypothermia has been one method used. Eastcott[4] reconstructed the carotid arterial flow after the body temperature had been reduced to 28°C. The flow was interrupted without use of systemic anticoagulants for 28 min. without obvious damage to the neural tissue. Boatman[1] and others have used hypothermia to protect the central nervous system from the effect of anoxia. Boatman also used, in conjunction with hypothermia, an intra-arterial bypass to resect an aneurysm of the internal carotid artery. This necessitated the use of systemic anticoagulants. Hypothermia, however, is time consuming and expensive, and its need during dissection of the neck may frequently not be anticipated, thus often making the equipment unavailable.

To minimize the interruption of arterial blood flow, Tibbs[10] devised a rather complicated and space consuming instrument. DeBakey and associates[3] and Cooley and co-workers[2] have described various shunts to maintain cerebral circulation, but they have also used systemic

anticoagulants to prevent clotting within this foreign body. Using the method described above, essential vessels infiltrated by tumor may be resected. A 1.5-cm. to 2-cm. residual segment of normal distal internal carotid artery is necessary. End-to-end reapproximation of the artery, anastomosis of the intact external carotid artery to the distal internal carotid artery, or graft replacement, may be used to re-establish cerebral blood circulation. The use of the bypass is also advised when re-anastomosis is possible without the interposition of a graft, since the method of anastomosis should be determined by the size and location of the vessel excised. As arterial invasion frequently follows invasion of the superior or middle jugular lymph nodes, this technique may often be employed. When an insufficient normal segment of the distal artery excludes the possibility of reconstruction, the fall in pressure within the distal internal carotid artery is measured after temporary occlusion. If the drop in pressure exceeds 50 per cent, ligation and excision of the diseased segment is probably unsafe. The arterial continuity should be re-established whenever possible, regardless of a small drop in distal carotid pressure after temporary proximal occlusion. The incidence of contralateral neoplastic spread and the coincidence of generalized and cerebral arteriosclerosis may threaten the adequacy of the flow in the one remaining carotid artery.

Lin[7] has re-emphasized the high complication rate following the insertion of foreign tissue or material in a widely dissected field which is subject to the accumulation of fluid, infection or delay in wound healing. These complications may be more evident following irradiation or in dissections that involve the oropharyngeal cavity. Embedding the graft or prosthesis in muscle allows good host acceptance and aids in excluding it from the areas which are heavily contaminated or infected.

SUMMARY

An inexpensive, simple, and easily prepared arterial bypass has been developed and described which allows the temporary maintenance of cerebral blood flow during resection of the carotid bifurcation or internal carotid artery until restoration of the circulation can be made by re-anastomosis or graft. Systemic anticoagulants were found to be unnecessary in the animal experiments and in the two clinical patients reported. The bypass transmitted up to 190 cubic centimeters of blood per minute when tested in laboratory animals. The technique of its successful use has been illustrated and described in two patients in whom neoplastic invasion necessitated excision of the carotid arteries. Its use is recommended during radical neck dissections in patients with neoplastic invasion of the wall of the carotid vessels.

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ACUTE POSTOPERATIVE PANCREATITIS

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The infrequency of postoperative pancreatitis lulls one into unawareness, but its lethal import should awaken us. Although it occurs rarely after distant operations (thyroidectomy, prostatectomy, normal delivery, and even blood transfusions), it is of major importance to general surgeons performing operations on the biliary tract, stomach, pancreas or spleen. The importance of postoperative pancreatitis is obvious when we realize that it contributes to one-half of the deaths after biliary tract surgery and to one-third of the deaths after gastric surgery.

ETIOLOGY

Unfortunately the cause of postoperative pancreatitis remains obscure. Suggested etiologic factors[14] include:

1. *Mechanical injury.* Operative trauma is apparently of some significance in that pancreatitis occurs more often after operations on the stomach, common duct or pancreas. However, it can occur when there has been complete absence or a minimum of pancreatic trauma; conversely, it often does not occur after rather severe operative manipulation of the pancreas.

2. *Vascular injuries.* The pancreas is such a vascular organ that it is unlikely that vascular injuries are of major importance in the usual case of postoperative pancreatitis. It is possible that an anomalous distribution of blood supply, particularly involving the superior and inferior pancreatoduodenals, may occasionally be of importance. As most cases occur after age 50, decreased vascularity from arteriosclerosis may be a contributory factor. The relative ischemia produced by shock during or immediately after surgery plus trauma may be of etiologic significance.

3. *Stagnation of duodenal contents.* This would be more prone to occur following Bilroth II gastrectomy and might allow regurgitation of infectious or chemically irritating material into the pancreatic ducts with resultant pancreatitis.

4. *Spasm and edema of muscles around the orifices of the pancreatic ducts.* Cholodochostomy with dilation and sphincterotomy would be particularly prone to produce a post-traumatic, edematous encroachment upon the pancreatic duct.

5. *Isolated lesions of the ductal system.* Millbourn[14] has emphasized that the duct of Santorini, which in 10 per cent of cases functions as the sole or chief excretory duct of the pancreas, is particularly vulnerable to damage or ligation during gastrectomy for duodenal ulcer as it enters the duodenum several centimeters proximal to the ampulla of Vater (fig. 1). Schmieden and Sebening observed 11 of 91 instances of acute pancreatitis complicating gastric surgery to be related to ligation of the duct of Santorini.

Other suggested causes for postoperative pancreatitis include sludging and increased viscosity of pancreatic secretions secondary to atropine, opiates, anesthesia and dehydration. Elliott and associates[9] recently presented excellent experimental studies making the common channel theory more acceptable, pointing out that pancreatic ductal pressure drops after 24 hours allowing reflux of pancreatic juice incubated in common duct bile into the pancreas with resultant acute pancreatitis.

DIAGNOSIS

Diagnosis depends essentially upon a high index of suspicion (an ingredient often lacking!) and early and repeated use of serum amylase determinations.

It is essential that we recognize the many limitations of serum amylase determinations so that blind faith will not be attached to their diagnostic significance.[8] Most are cognizant of the rapid rise of serum amylase in acute pancreatitis will return to normal in 48 to 72 hours, making it obligatory that *early and repeated determinations* be made in any suspected case. It is also appreciated that a fulminating pancreatitis may be associated with such severe necrosis that serum amylase determinations will be normal, only minimally elevated or actually subnormal. In general, one is unable to correlate the degree of hyperamylasemia with the severity of pancreatitis.

Preoperatively the limitations of serum amyl-

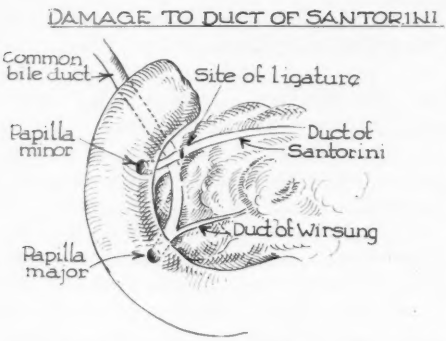

FIG. 1. Depicts vulnerability of duct of Santorini to damage or ligation during gastrectomy for duodenal ulcer (after Millbourn).

ase values must be borne in mind in assessing any acute abdomen. It has been repeatedly shown that acute cholecystitis without pancreatitis, ruptured peptic ulcer (especially larger perforations occurring within a few hours after a meal), high intestinal obstruction and mesenteric venous thrombosis can on occasion cause a markedly elevated serum amylase, often over 1,000 units. *If the clinical findings indicate laparotomy in any acute abdomen, the elevated serum amylase should not deter one!*

Significant elevations of amylase (increases of over 100 per cent) occur in 10 to 15 per cent of patients having subtotal gastrectomy or exploration of the common duct. Millbourn found significant urinary diastase elevation 4 times more frequently in patients having gastrectomy for duodenal ulcer as compared to gastric ulcer or carcinoma (17 per cent : 4 per cent). Although many of these patients probably do have mild or edematous pancreatitis, it is now realized that a significant number of patients receiving opiates, particularly morphine and codeine, have appreciable elevations in serum amylase. This is no doubt the mechanism of many lesser elevations of serum amylase in the postoperative period.

Despite the aforementioned limitations, early and repeated serum amylase determinations can be of utmost importance in establishing a diagnosis of postoperative pancreatitis. This should be routine in all patients with pancreatic trauma and in patients with unexplained abdominal signs and symptoms. Later in the disease, the development of hypocalcemia or hyperglycemia are of diagnostic aid.

Paracentesis with examination of peritoneal fluid can be of great value in the diagnosis of postoperative pancreatitis. It has been shown that the peritoneal fluid amylase is more elevated and lasts 2 to 4 days longer than does the serum amylase. It should be emphasized that necrotic intestine is often associated with a peritoneal fluid amylase exceeding 1000 units, apparently due to leakage of intraluminal enzyme through the abnormally permeable intestinal wall.[12] An immediate Gram stain of the peritoneal fluid is the best way of differentiating pancreatitis from infarcted intestine, for gram-negative bacilli will usually be found in the latter.

CLINICAL PICTURE

Although there are no pathognomonic manifestations of postoperative pancreatitis, the possibility should be strongly considered after upper abdominal surgery (particularly gastrectomy, common duct exploration or sphincterotomy) when any of the following conditions present:

1. *Severe, persistent, unyielding pain*, greater than would be expected from the operative procedure and often refractory to usual dosage of narcotics.

2. *Vascular instability or shock.* A labile blood pressure, unexplained hypotension and increasing tachycardia are often the first indications of the complication.

3. *Persistent ileus* with distention, nausea, and vomiting often occurring despite gastric suction.

4. *Profuse drainage* of bile-tinged or serosanguineous material by means of rubber tissue drains. The use of such drainage as a routine is essential following common duct surgery or difficult inversion of a duodenal stump.

5. *Unexplained anemia.* Significant drop in the hematocrit (5 to 10 mm.) without obvious blood loss often heralds postoperative pancreatitis.

6. *Hiccoughs.* Intractable singultus, apparently due to diaphragmatic irritation from pancreatic enzymes, can be an important clue.

7. *Low grade fever.* Although more difficult to assess, low grade fever commonly occurs with pancreatic necrosis.

8. *Oliguria* and renal shut-down not infrequently are secondary to this postoperative complication.

9. *Mild jaundice*, often subclinical, due to encroachment on the common duct by the in-

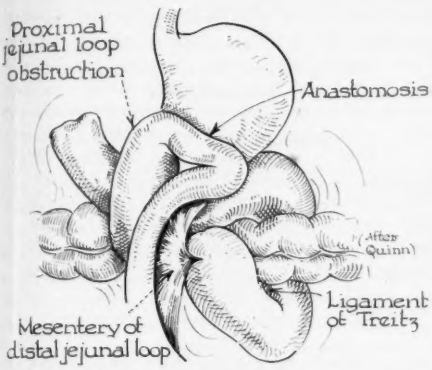

FIG. 2. Afferent loop obstruction is more prone to follow antecolic anastomosis and closely simulates postoperative pancreatitis.

volved head of the pancreas or to absorption of extravasated bile from the peritoneal cavity.

10. *Left pleural effusion*, due to the rich lymphatic interchange through the diaphragm.

11. *Palpable abdominal mass* secondary to pancreatic necrosis and/ or infection.

12. *Hyperglycemia.*

DIFFERENTIAL DIAGNOSIS

When there is diagnostic doubt, it will often become necessary to explore the patient, particularly after gastric resection, for the following conditions closely simulate acute postoperative pancreatitis and are often associated with significant elevations of serum or peritoneal fluid amylase:

1. *Afferent loop obstruction* (fig. 2). This is more likely to occur with a long-loop, antecolic anastomosis. It is associated with severe epigastric or left upper quadrant pain and tenderness attended with clinical indications of shock. It should be suspected when there is limited vomiting, with absence of bile and small bowel contents in the vomitus. One may feel the distended bowel as a mass in the left upper quadrant. As no air enters the obstructed loop, x-ray will show no evidence of gas-filled, distended bowel. Entero-anastomosis between the distended and collapsed jejunal loops is mandatory to avoid perforation and death.[16]

2. *Blow-out of the duodenal stump*, occurring in 2 to 3 per cent of gastrectomies for duodenal ulcer, represents a true surgical emergency unless the blow-out has been anticipated and appropriate sump drain placed down to the stump.

3. *Mesenteric venous thrombosis* is fortunately quite rare after upper abdominal surgery but closely simulates acute pancreatitis. Significant peritoneal fluid findings include its cloudy reddish brown appearance with mild fecal odor, markedly elevated amylase (usually greater than 1000 units) and presence of *Escherichia coli.* Immediate Gram stain searching for *E. coli* is imperative as their presence denote intestinal infarction and demand immediate surgical intervention.

4. *Delayed splenic rupture.* Although usually appreciated at the time of gastrectomy, delayed splenic hemorrhage upon rare occasions follows gastrectomy. As it may be associated with an elevated serum amylase, each case must be carefully evaluated clinically.

OPERATIVE PRECAUTIONS

One should treat the pancreas as a powder-keg capable of devastating explosion[14] and limit operative trauma to an absolute minimum. Zollinger's statement that one should dilate the ampulla of Vater with the same gentleness and temerity that would dilate his own urethra is worthy of emphasis. Rarely will it be necessary to dilate the sphincter in excess of 5 mm. If difficulty in passing a Bakes dilator into the duodenum is encountered, the attempt should be abandoned, and resort made to immediate cholangiography or to transduodenal visualization of the ampulla of Vater with or without sphincterotomy. If sphincterotomy is done, sutures about the ampulla are to be avoided for they may unknowingly encroach upon the pancreatic duct. Common duct irrigation must be gentle as it has been repeatedly shown experimentally that forceful injection of bile into pancreatic ducts uniformly results in acute pancreatitis. The use of long T-tubes or catheters, even with perforations, passed through the ampulla of Vater is to be avoided as there are numerous documented cases of postoperative pancreatitis apparently induced by this mechanism. There is often such a thin septum between the common bile and pancreatic ducts that such tubes may occlude, partially or completely, the pancreatic duct by pressure on the septum (fig. 3).

There are occasional cases of massively penetrating duodenal ulcers which should be treated primarily by vagotomy and posterior antral gastrojejunostomy for the risk of pan-

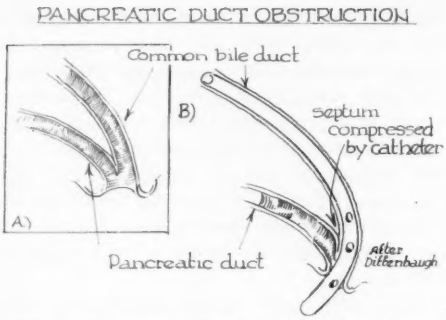

FIG. 3. Long-limb T-tubes or catheters passed through the ampulla of Vater are capable of encroaching upon the pancreatic duct with resultant severe pancreatitis.

creatitis and leakage of the duodenal stump outweigh the 8 to 10 per cent incidence of recurrent ulceration attendant to this procedure. In penetrating gastric or duodenal ulcers it is essential that the stomach or duodenum be gently separated from the ulcer bed, which should neither be excised, cauterized, curetted, or oversewn! In the case of duodenal ulcer, however, it should be carefully inspected to be sure that there is not an open pancreatic duct in the ulcer base. Cognizance of the vulnerability of the duct of Santorini in surgery of duodenal ulcer is imperative; ligation can be harmless, but may be disastrous. Occasionally an "open duodenum" technique allowing palpation of the papillae of Vater and Santorini will be useful. The duodenum should not be freed from the pancreas closer than 3 cm. proximal to the greater duodenal papilla.

TREATMENT

Although it is admitted that exploratory laparotomy is often necessary to establish the diagnosis of acute postoperative pancreatitis, treatment, when feasible, should be conservative in that there is no definitive operation for acute pancreatitis. Meperidine is the drug of choice for pain relief in that it produces less spasm of the ampulla of Vater. Continuous epidural block is valuable in relieving intractable pain. Minute attention must be paid to fluid and electrolyte balance with frequent blood chemistries being essential in correcting sodium, potassium and chloride deficits. Blood in large amounts is indicated early in the disease for there is often more of a reduction in the blood volume than can be appreciated by red blood count or hematocrit determination. Pancreatic secretory activity should be suppressed with probanthine and continuous gastric suction, for gastric acidity in contact with the ampulla of Vater is a powerful pancreatic secretagogue. Prostigmine is contraindicated because of its stimulatory effect to pancreatic secretion. Liberal use of antibiotics to combat infection is necessary. The use of corticords is debatable, but on occasion has averted what would have otherwise been a fatal outcome.

TABLE 1

Analysis of cases of postoperative pancreatitis observed during the last decade at 3 local hospitals

Procedure	No. of Cases	No. of Deaths
Gastrectomy	5	0
Common duct procedure	7	3
Pancreatectomy	1	0
Nephrectomy	1	0
Total	14	3

ANALYSIS OF CASES

In the 1950 to 1960 decade, there has been 14 unequivocal cases of postoperative pancreatitis in the 3 local hospitals (Mississippi Baptist, St. Dominics, and University Hospitals) with 3 deaths, a mortality of 21.4 per cent (table 1). No doubt, many of the milder cases recovered without diagnosis; too, improved autopsy rate would have unquestionably revealed other completely unsuspected cases.

Of these 14 cases 6 were clinically unappreciated and not discovered until operation or autopsy; in 2 others the diagnosis was considered only quite late. Certainly an increasing awareness of this potentially lethal postoperative complication is needed!

Postgastrectomy pancreatitis. Although there was no mortality in the 5 postgastrectomy cases, the morbidity in 2 was marked. Two were re-explored with a diagnosis of leaking duodenal stump:

Case 1. On the 1st postoperative day after subtotal gastrectomy for a chronic duodenal ulcer penetrating into the pancreas, the patient presented severe diffuse abdominal pain and

profuse drainage of thin, bile-tinged fluid along the drain placed down to the inverted duodenum. Exploration showed severe pancreatitis with clear pancreatic and bile-tinged fluid oozing from the entire pancreatic head. Cardiac arrest was effectively combatted with massage; the pancreatic area was generously drained with sump and rubber tissue drains. The daily sump drainage varied from 500 to 2000 cc. for the next 10 weeks. As the patient was steadily losing ground because of the profuse unrelenting drainage, he was reexplored. Free bile was draining from a necrotic distal common duct. The common duct was deliberately ligated and cholecystojejunostomy effected. After this procedure pancreatic drainage continued for an additional 3 months but eventual recovery ensued. The possibility of acute pancreatitis being associated with necrosis and perforation of the common bile duct has been reported by others. ([4, 20]). *Although heroic, deliberate ligation of the common duct with cholecystojejunostomy without question precipitated recovery in this gravely ill individual.*

Case 2. Following three-fourths subtotal gastrectomy for chronic duodenal ulcer, there was completely smooth postoperative course with discharge on the 5th postoperative day. The patient celebrated his smooth recovery with a 3-day alcoholic bout and returned to the hospital on the 12th postoperative day with severe upper abdominal pain. Pancreatitis was strongly suspected here; operation became necessary in 36 hours when there was increase in abdominal pain, tenderness and fever suggesting duodenal stump leakage. The stump was intact, however, and the pathology was that of acute pancreatitis with fat necrosis. Recovery with drainage ensued although vasopressors were necessary for 48 hours to maintain blood pressure.

Another postgastrectomy pancreatitis was manifest by obstruction of the efferent jejunal loop. Recovery after insertion of a gastrostomy tube for drainage and a jejunostomy tube for feeding ensued.

Four hours after hemigastrectomy and vagotomy (in which there had been minimal pancreatic trauma), one patient presented a labile blood pressure, rising pulse rate with a drop in hematocrit. Serum amylase was 804 units. Recovery with vigorous conservative therapy occurred promptly although free fluid was evident in the abdomen.

Pancreatitis after common duct procedures. Six of the cases followed choledochostomy; one followed sphincterotomy with choledochoduodenostomy. Two were associated with the use of longarm T-tubes extending through the ampulla of Vater. Two were manifest by prolonged febrile course, before developing palpable masses requiring incision and drainage. One of these was diagnosed by hypocalcemia (3.6 mEq. per l.).

It is disconcerting to find that in two cases (one terminating fatally), the common duct was explored with minimal indication. In both the cystic and common ducts seemed to be of normal size; choledochostomy was done because of small stones in the gall bladder in 1 case and because of "long-standing" gall bladder disease in the other. Although the need for exploring the common duct is obvious to all surgeons, these 2 cases would emphasize that one should not be indiscriminate about such exploration. In my experience the size of the cystic duct has been the most reliable single criterion as to the presence of stones in the common bile duct. *If multiple small stones in the gall bladder are associated with a small cystic duct and a common duct of normal size, it is doubtful that choledochostomy should be done.*

One episode of acute pancreatitis with elevation of serum amylase to 1330 units occurred when an elderly male inadvertently pulled the T-tube out as he changed position in bed. This further emphasizes the need for gentle T-tube manipulation. A number of years ago in attempting to remove a retained common duct stone by irrigation with ether-alcohol mixture, a rather severe hemorrhagic pancreatitis was produced. The questionable results to be obtained with such ether-alcohol irrigations and the danger of acute pancreatitis make this a procedure of doubtful virtue. Though invaluable, T-tube cholangiograms must be done gently for pancreatitis can be precipitated thereby. One fatal case of pancreatitis after simple T-tube cholangiography has been reported!

SUMMARY

The numerous nondescript, but occasionally bizarre manifestations of postoperative pancreatitis are emphasized to increase the awareness of this relatively infrequent, but catastrophic, postoperative complication. Minimizing operative trauma particularly in operations on the common duct and stomach is specifically emphasized. Major diagnostic clues include pain out of pro-

portion to that expected from the operation itself, vascular instability and shock, persistent ileus, profuse drainage and the development of unexplained anemia.

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RECURRENT MITRAL STENOSIS AFTER MITRAL VALVOTOMY SURGICAL CONSIDERATIONS*

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Recurrence of mitral stenosis following mitral valvotomy is not rare. Indeed, with the rapidly increasing number of operations on the stenotic mitral valve being performed, hundreds of patients may appear in the near future with return of symptoms and findings of mitral stenosis.[1] Fortunately, a true recurrence of mitral stenosis after a technically satisfactory valvotomy is uncommon and is exceedingly gradual in its progression unless rheumatic fever occurs after operation. Therefore, operation in relatively young patients and those with known rheumatic activity should be avoided. Mitral stenosis following an incomplete valvotomy is usually referred to as recurrent although the obstruction at the valve was not relieved initially. Among the factors which lead to failure of a complete valvotomy are calcification of the commissures, elasticity of the fibrous tissue and fusion of chordae tendineae, extensive intra-atrial thrombosis, arrhythmias, etc. Occasionally a surgeon of limited experience may be hesitant to proceed under difficult conditions. Although many patients obtain an initial improvement when such factors exist, often benefit is limited in degree and duration and manifestations of stenosis soon recur.

For a time in the past we utilized the pump oxygenator and temporary cardiopulmonary bypass for cases of recurrent mitral stenosis. This method of valvotomy was complicated, requiring extensive preparations, and imposed certain hazards to the patient associated with the technique of bypass. In 11 patients treated by the open technique, 1 fatality occurred from pulmonary embolism 10 days after operation.[2] Ultimate functional results in these patients were good although the initial morbidity was high when compared to closed techniques. Cerebral air embolism occurred in 4 patients rendering them unconscious for 24 hr. to 5 days. All of these patients recovered after a stormy course during which convulsive seizures were frequent. Postoperative bleeding in the heparinized patient was another troublesome complication. The technical repair of the stenotic valve under direct vision was disappointing, and often a dilation of the valve with a large hemostat provided the most satisfactory means of achieving the desired result without producing mitral insufficiency. The principal advantage of the open operation in our opinion was that it permitted complete removal of all contained intra-atrial thrombi and prevented embolism of solid material. In general, however, these results were disappointing, and more recently techniques of closed valvotomy have been used routinely with certain important modifications.

* From the Cora and Webb Mading Department of Surgery, Baylor University College of Medicine and the Methodist, Jefferson Davis, St. Luke's and Veterans Administration Hospitals, Houston, Texas. Supported in part by the United States Public Service under Grants H-3137 and HTS-5387, the S. J. Thibodeaux Foundation, and Houston Heart Association.

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TECHNICAL CONSIDERATIONS

Logan[5] of Edinburgh has demonstrated the feasibility of a transventricular technique of mitral valvotomy and recently reported on 438 operations done by this method. He considered the results of this technique superior to any others previously used. Approximately 2 yr. ago we began using this method almost routinely in mitral valvotomy.[3] Since then we have performed 45 transventricular valvotomies on patients undergoing their initial operation. Functional results in our cases were better than results of operation by finger fracture or cutting valvotomes as used previously.[2] Minor modifications of the transventricular technique have been made for application to cases of recurrent mitral stenosis. Two approaches to the valve are now employed depending upon whether atrial thrombosis is known to be present from the previous operation. If thrombosis around the left atrial appendage is not suspected, a unilateral left-sided approach is used. For cases of suspected thrombosis in the appendage a bilateral approach is used.

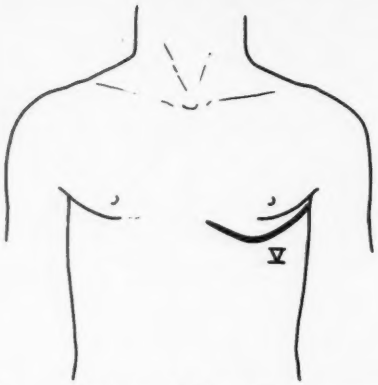

FIG. 1. Drawing showing left submammary 5th intercostal space incision used in unilateral left approach to valvotomy for recurrent mitral stenosis.

FIG. 2*a*. Drawing showing method of control of hemorrhage in digital exploration of left atrium. Mattress sutures of stainless steel are inserted and the area of entry is surrounded with a heavy purse string suture. *b*. After the finger is withdrawn from the heart, the atriotomy incision is repaired with interrupted silk sutures, and the mattress and purse string sutures may be removed.

UNILATERAL LEFT APPROACH

The patient is placed in the supine position with his left shoulder slightly elevated; the surgeon stands on the patient's left. An anterior 5th intercostal space incision is made without dividing costal cartilages (fig. 1). Adhesions between lung, parietal pleura and pericardium are divided, and the pericardium is incised anterior and parallel to the phrenic nerve. A pliable area on the lateral aspect of the left atrium is selected for the atriotomy incision. A technique recommended by Kay[4] for entering the side of the atrium is used. Four mattress sutures of braided wire are inserted in the atrial wall, and the ends of the suture are held individually with small hemostats (fig. 2*a*).When the incision is made in the atrium, the right index finger is inserted between these sutures and bleeding is controlled by making traction on the suture. For additional control a heavy purse string suture is placed about the entire area enclosed by the mattress sutures. A Rumel cardiac tourniquet with ratchet handle is convenient for control of the purse string suture since once the ratchet is set the surgeon may release the handle of the instrument. Wire sutures are useful since they will not be divided when the atriotomy incision is made. After the finger is withdrawn, the sutures are pulled up controlling the bleeding while the atriotomy is repaired with silk sutures (fig. 2*b*). The mattress and purse string sutures may then be removed. The presence of scarring of the atrium assists in the control of bleeding since sutures have less tendency to pull out of the heart.

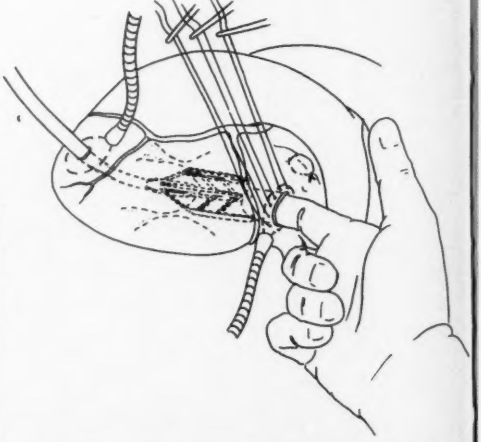

FIG. 3. Drawing showing transventricular valvotomy being performed from unilateral left-sided approach. With the right index finger inserted in the left atrium the expanding dilator is introduced through the valve orifice from the ventricular apex. The valvotome is opened to 4.0 to 4.5 cm. width. (From George P. Pilling & Son Company, Philadelphia, Pennsylvania.)

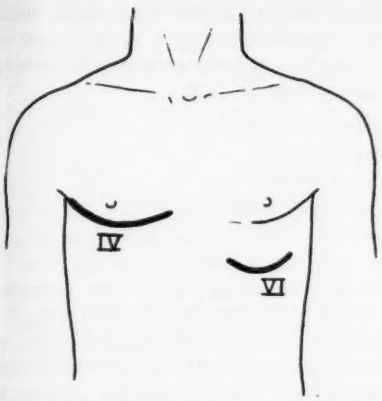

FIG. 4. Drawing showing incisions used in bilateral approach to mitral valvotomy using transventricular technique for recurrent stenosis. Right 4th intercostal space and smaller left 6th space incisions are used without dividing costal cartilages.

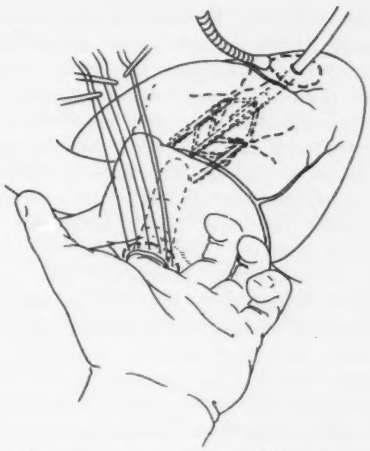

FIG. 5. Drawing showing technique of transventricular mitral valvotomy using bilateral approach. With the left index finger inserted in the mitral orifice the expanding dilator is inserted into the valve through the apex of the left ventricle.

Before the valve is explored, a purse string suture of heavy silk is placed at the apex of the left ventricle in an area which has relatively few coronary vessels on the surface. An incision is made through the epicardium and enlarged with the tip of a hemostat. With the right index finger inside the left atrium palpating the valve orifice from above, the expanding dilators are inserted from below (fig. 3). The dilators are guided into position avoiding entanglement with papillary muscles and chordae tendineae. The valve orifice is dilated to the desired size, usually approximately 4.0 cm. in length providing effective relief of stenosis. Mitral incompetence following this technique is surprisingly uncommon particularly when such an excellent relief of stenosis is achieved. Upon completing the valvotomy, the atriotomy and ventriculotomy are repaired with silk sutures. Usually the procedure is associated with slight blood loss, and blood transfusion is not necessary. The thoracotomy incision is closed with underwater drainage.

BILATERAL APPROACH

The patient is placed in the supine position on the operating table; the surgeon stands on the patient's right side. An anterior 4th intercostal space incision is made without dividing costal cartilages (fig. 4). The pericardium is incised parallel to the phrenic nerve, and the interatrial groove is exposed by gentle traction on the overlying right atrium. Wire mattress sutures and an encircling purse string suture of heavy silk are inserted in this area similar to the method used for entering the left atrium in the left unilateral approach. A short anterior curved incision is made in the left 6th intercostal space for exposure of the left ventricle. The pericardium is incised, and a heavy purse string suture is inserted about the apex of the ventricle. The operator then makes a left atriotomy incision and inserts his left index finger for exploration of the valve. The transventricular mitral dilator is then inserted through the left thoracotomy incision and is manipulated by the surgeon's right hand (fig. 5). Valvotomy by the bilateral approach is performed in the manner already described for transventricular valvotomy. Upon completion of the valvotomy, the atriotomy and ventriculotomy are repaired with silk sutures. The pericardium is closed loosely, and the thoracotomy incisions are closed using bilateral underwater drainage.

RESULTS

Transventricular valvotomy for recurrent mitral stenosis has been utilized in 17 patients without a death or serious complication. Three patients have evidence of mild mitral incompetence of minor significance. In 12 instances a unilateral left approach was used and in 5 a

bilateral approach. The bilateral approach using separate incision has been better tolerated than the method of transecting the sternum as previously described by us.[3] In the bilateral approach the dense adhesions which may be present around the left atrium may be avoided, thus simplifying the dissection and providing another advantage of this technique over the unilateral approach.

Superior functional results have also been obtained when the transventricular incision was used for an initial valvotomy. Two deaths occurred among 45 patients. One of these died from mitral incompetence following extreme and excessive dilation of the valve in an early case. Another patient died suddenly on the fourth postoperative day from a large ball valve thrombus which occluded the mitral orifice 3 days after operation.

SUMMARY

Surgical treatment of recurrent mitral stenosis can be satisfactorily accomplished by a closed procedure. Transventricular valvotomy using a unilateral left or bilateral thoracotomy usually provides excellent relief of stenosis with low mortality. This technique was used in seventeen patients without a death.

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SEMINAR ON KIDNEY DISEASE

The Southeastern Region of the College of American Pathologists and the Virginia Society of Pathologists will hold a joint meeting at the John Marshall Hotel in Richmond, Virginia, on November 25 and 26, 1960, on kidney disease. The speakers will include Drs. Stanley M. Kurtz, Peter P. Ladewig, Henry D. McIntosh, George Margolis, Conrad L. Pirani, David E. Smith, and Max Wachstein. The slide seminar will be conducted by Drs. Paul Kimmelstiel and Solomon Papper. The dinner speaker will be Dr. Frank C. Coleman, president of the College of American Pathologists.

The slide sets for this seminar on kidney disease may be purchased at a cost of $15.00 per set by writing to: Dr. G. T. Mann, Professor of Forensic Pathology, P. O. Box 41, Medical College of Virginia, Richmond 19, Virginia.

SURGICAL MANAGEMENT OF THE PATIENT WITH UNDIAGNOSED MASSIVE UPPER GASTROINTESTINAL TRACT BLEEDING

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One of the most challenging problems to confront the surgeon is the patient vomiting bright red blood. Such massive bleeding usually denotes hemorrhage proximal to the ligament of Treitz. Even with this arbitrary exclusion of ⅚ of the gastrointestinal tract, localization of the bleeding point preoperatively and during exploration is notoriously difficult. Massive upper gastrointestinal tract bleeding is an emergency which does not permit leisurely evaluation of the patient, and even if it did, the procedures which are currently available do not assure precise definition of the source of hemorrhage in all patients. Indeed the origin of bleeding may not be found at operation or even at postmortem examination.[2]

The records of all the patients presenting themselves with massive hematemesis at the University Hospital and the Veterans Administration Hospital, Birmingham, for the past 5 years (1954 to 1959) have been reviewed. Only patients who were actually observed to vomit bright red blood or who had bright red blood in the gastric aspirate were included in this report. There were 232 such cases. Of these 232 patients, 60 had emergency exploration. No patient scheduled formally 12 hr. before the operation was considered an emergency, and most of these operations were performed in the early morning hours. In 52 patients there was a positive diagnosis of the bleeding lesion at the time of operation (table 1).

An analysis will be made of the validity of the diagnoses before operation, identification of lesions at operation, and the mortality rate. We shall also consider the patients in whom no lesion responsible for bleeding was found at operation (table 2). The incidence of re-bleeding in this group after "blind" gastrectomy, or no definitive procedure will be tabulated. Peptic ulcer will be included for statistical purposes, but not discussed primarily, since this lesion is more easily identified before, or at the time of operation. Methods employed to control bleeding in the preoperative period will be reviewed as well as procedures for identification of the bleeding site. Operative aids in localization of the bleeding lesion at the time of surgery will be discussed.

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EMERGENCY MANAGEMENT

Treatment of Shock

No other condition except a blocked trachea requires more rapid treatment than shock. This should take precedence over any other phase of emergency management. Preferable to the futile attempts at venipuncture with its delays and discomforts is the direct exposure of a suitable vein and the placement of a polythene cannula which will facilitate the restoration of an adequate blood volume. Specimens of blood necessary for cross matching and typing as well as indicated laboratory tests can also be secured through the cannula. Appropriate solutions should be started immediately. Then, preferably within the hour, fresh whole blood should be administered in quantity sufficient to restore vital signs to normal. The blood should be as fresh as possible, and should have been collected in plastic bags to avoid platelet attrition[18] and the increased ammonia content of stored blood which may initiate coma if the patient has intrinsic liver disease.[3]

INTUBATION

It is our practice to advocate the use of a tube in all patients with massive hematemesis. The tube should be placed before any diagnostic procedure. Unless there is irrefutable evidence of peptic ulcer, from old charts or from the referring physician, or a responsible relative, our personal choice is to use the double balloon tube designed for tamponade of the lower third of the esophagus.[23] It does not require x-ray monitoring for accurate placement. It will satisfactorily evacuate blood and clot from the stomach. It lessens respiratory difficulty by arresting vomit-

TABLE 1

Lesions Found at Operation	No. of Cases
Peptic ulcer (gastric and duodenal)	32
Varices	6
Carcinoma stomach	1
Rare lesions	
Mallory-Weiss syndrome	1
Lymphosarcoma stomach	2
Leiomyoma stomach	1
Liposarcoma stomach	1
Carcinoid duodenum	1
Metastatic melanoma with erosion of duodenum	1
Carcinoma of pancreas with erosion of pancreato-duodenal artery	1
Pancreatic abscess with gastritis	1
Pylephlebitis with erosion of hepatic artery	1
Arteriovenous aneurysm jejunum	1
Acute cholecystitis with erosion of cystic artery	1
Reticulum cell sarcoma spleen	1
Total	52

TABLE 2

Sixty patients operated on for massive upper gastrointestinal hemorrhage

	No. of Cases	Percentage
Correct diagnosis preoperative	37	61.6
Undiagnosed before exploration	23	38.3
Diagnosis at operation	16	69.5
No diagnosis at operation	7	30.5

ing. When these things have been accomplished, a more accurate appraisal of blood loss is possible. Inflation of the balloons and observation of the gastric aspirate make a differential diagnosis possible. If the gastric aspirate clears, one may assume that the bleeding has its source in the lower third of the esophagus. If the aspirate continues to be bloody, bleeding is localized to the stomach or the duodenum and should suggest ulcer or gastritis. The one exception, however, is gastric varices which are present in 2 per cent of cirrhotic patients.[23, 26] If the bloody gastric aspirate continues, the balloons are deflated, and the tube is used as a Levin tube. It can then be employed to keep blood out of the stomach before operation, or if the bleeding stops, it can be used for feeding, intestinal antibiotics, cathartics, or the institution of the buffer and Thrombin regime.

If a bloody gastric aspirate continues, further information may be gained by the use of buffer and thrombin.[6, 7] If the bleeding is diffuse, (gastritis or shallow ulceration) buffer solution is injected through the tube into the stomach, and the tube is clamped for 2 minutes. This enables a shift in pH at which Thrombin will not be inactivated. The tube is re-opened and 10,000 units of topical Thrombin is injected through the tube, and the tube is again clamped for ½ hour. This method should not be blindly relied upon, but may be used during the period of evaluation. If bleeding is not controlled promptly, one should assume that an eroded vessel is the cause of hemorrhage and abandon this regime. This method was not designed to control bleeding from an eroded vessel, and will not do so.

After these emergency measures have been accomplished attempts at diagnosis with indicated diagnostic procedures can proceed.

DIAGNOSTIC PROCEDURES

History

Every effort should be made to secure an adequate history from the patient. Bleeding tendencies, medications, alcoholic ingestion and previous admissions to the hospital are important. Old charts should be secured whenever possible. The patient's condition often does not permit lengthy cross examination, and information elicited from distraught relatives is often misleading and erroneous.

Physical Examination

A rapid but thorough physical examination should be carried out, preferably by the responsible physician. These patients tolerate repeated examination poorly. It is important to examine the back of the patient as well as the front. One should be alert for the stigmata of cirrhosis, telangiectasias of the mucous membranes, and purpuric spots. Of equal importance are areas of abdominal tenderness and splenic enlargement without accompanying hepatomegaly. Indeed this may be the only clue to extrahepatic portal hypertension which can be found especially in children and young adults.[12] An audible venous hum may also indicate extrahepatic portal hypertension (Cruveilhier-Baumgarten's Syndrome).

Laboratory Procedures

Careful correlation of information obtained from the history and physical examination will obviate many exhaustive and time consuming laboratory examinations. These studies should evaluate resuscitation from shock, continuation or cessation of bleeding, and the status of liver function.

It is vital to have routine hematocrit and hemoglobin levels, with blood smears to determine the adequacy of platelets. Urine should be examined for constituents and a recording of hourly volume should be made to estimate success in restoring a normal blood volume. A stool guaiac examination is important because of the information it provides about the duration of the bleeding episode. Stools will be negative if the bleeding has been of short duration.

Liver functions can be adequately assessed by 3 procedures, and may indicate the basic cause of the hemorrhage. The test most readily obtained in any hospital is the prothrombin time. Patients bleeding massively from peptic ulcer or other gastrointestinal lesions will have no prolongation of the prothrombin time. Patients with underlying liver disease will have markedly prolonged prothrombin times, and esophageal or gastric varices are therefore suspect. The exception to this, of course, is the patient with extrahepatic obstruction with varices. Since the liver is normal there will be no alteration of the prothrombin time. This group is usually composed of children and young adults.

The Bromsulphalein test is carried out in the usual fashion if jaundice is not present. After 30 min. a retention of dye to the amount of 10 per cent or less is considered normal. Marked dye retention will again focus attention on liver disease.[13, 31] This test may be misleading because of the association of peptic ulcer with liver disease which occurs in 15 to 20 per cent of all patients with cirrhosis.[8, 14, 20, 29]

The other test which has frequently been of value but is not performed in all hospitals is the blood ammonia determination. This test indicates poor liver function if elevated (normal 48 to 72 gamma per cent), but again it must be interpreted cautiously since elevations of ammonia by freshly shed blood into the upper bowel does occur when no liver disease is present.[17]

In short, all of these tests provide help in the diagnosis but may also be misleading. It is necessary to correlate them with the history and the physical findings.

Emergency X-ray Examination

There has been increasing interest in emergency barium or gastrografin studies for the patient with massive hemaetemsis.[25] These studies, if performed, should be carried out when shock has been successfully treated and vital signs have returned to normal. Manipulation of the poorly stabilized patient is fraught with hazard. When positive information is obtained it is a definite advantage. Interpretation, however, is rendered difficult in the presence of blood and clots. During a major bleeding episode distended varices will collapse. These may be demonstrated in about 60 per cent of the patients examined. Chronic peptic ulcers can be demonstrated in about 90 per cent of examinations performed, but acute ulcerations are seldom ever seen. This procedure may be tried but too much reliance should not be placed on the interpretation.

Splenic Pulp Pressures

Recently the measurement of splenic pulp pressures percutaneously has been advocated in the emergency and elective assessment of portal hypertension.[1, 21, 30] It should only be carried out when the spleen is palpable, when the prothrombin level is above 60 per cent, when no bruising is present, and when the patient is able to cooperate. This is often advantageous in the patient with normal hepatic studies in whom extrahepatic portal hypertension is suspected. We have not obtained consistent correlation between levels of pressure in the splenic pulp and in the portal vein by direct measurement. It may well be that our experience is not sufficiently great, but during an episode of massive hematemesis there is decompression of the portal bed by hemorrhage from the bleeding varix. Confusion in interpretation ensues. It would, however, seem to merit a further trial.

Endoscopy

The use of the esophagoscope, the flexible gastroscope, or the new Fiberscope*, [5, 10] may

* American Cystoscope Makers, Inc., New York, New York.

localize the bleeding area by direct inspection. These instruments can be used at the bedside, and the examination should be carried out in the unanesthetized cooperative patient. The Fiberscope will also permit scrutiny of the duodenum. The presence of blood and clot in the stomach even after attempts at their removal makes examination by this means difficult.

DECISION FOR OPERATIVE INTERVENTION

After the foregoing measures have been expeditiously carried out decision for further management will have to be made. If the vital signs have stabilized and the bleeding has stopped, continued study and observation of the patient may be in order. If the vital signs have not stabilized or precarious stability is achieved only by the administration of large amounts of whole blood (more than 500 cc. per hour), massive continuing hemorrhage will dictate early operation regardless of the prior establishment of a diagnosis. The criteria dictating emergency surgery at our institution are as follows:

1. *Failure to achieve stabilization of vital signs after massive blood replacement.* By massive blood replacement we mean 1000 to 1500 cc. whole blood given in 1 to 2 hr. This may be evaluated by pulse blood pressure and the hematocrit.

2. *Re-bleeding after initial control of bleeding.* With the Levin tube in place the gastric aspirate is constantly observed. The appearance of bright red blood in the aspirate does not prompt us to go through another period of resuscitation, but the patient is taken to the operating room within the hour.

3. *Age.* When advancing age is complicated by artherosclerosis early operation is mandatory. These patients respond poorly to a period of cerebral, myocardial or renal ischemia. If the operation is unduly delayed some survive the procedure but die in the postoperative period of myocardial infarction or renal failure. The eroded atherosclerotic vessel in the base of an ulcer crater can only be controlled by ligature.

4. *Availability of blood.* Large amounts of whole blood, especially the rarer types, are hard to come by even in a large institution. While it is not the major consideration, it must play an important part in the decision for prompt surgical intervention.

5. *Administration of large amounts of whole blood.* If large amounts of correctly cross-matched and typed whole blood are available there is a temptation to delay early operation for what may seem to be valid considerations. The hemorrhagic states induced by rapid administration of large amounts of whole blood are well known and may further complicate the desperate circumstances already present.[11]

There is no substitute for constant observation of these patients and impressions so gained should dictate management to be employed, taking precedence over any information gained by laboratory or diagnostic procedures.

OPERATIVE MANAGEMENT

Once the decision is made for operative intervention adequate supplies of fresh whole blood must be available. An x-ray casette should be placed in the operating table so that films may be made during the operation if desired.

1. *Anesthesia.* We have used general anesthesia administered by the endotracheal route. Regardless of one's personal convictions concerning the use of a tube in these bleeding patients, a tube should be passed into the stomach before induction and kept on constant suction. This will prevent vomiting and the aspiration of blood and clot during induction and in the immediate postoperative period.

2. *Incision.* The incision to use is a matter of personal choice and should be the one with which the surgeon is most familiar. Whether or not the source of bleeding is diagnosed before operation we use the left subcostal incision which can easily be extended to the right subcostal area. This approach provides a rapid and easy inspection of the various organs and structures to be examined. It is obvious that this must be a systematized examination if aimless and time-consuming muddling is to be avoided.

Operative Procedure

A. Inspection. There are some lesions which are identifiable by inspection of the viscera. The scarred duodenum suggests duodenal ulcer. The contracted rigid stomach will suggest malignancy. The hob-nailed liver will call attention to the possibility of portal hypertension. The area of conglomerate telangiectasia is easily seen. It is important to note any dilated omental veins or dilated capillaries coursing over the antimesenteric side of the duodenum or jejunum. Careful

TABLE 3

Lesions Found at Operation by Inspection	No. of Cases
Peptic ulcer (gastric and duodenal)	28
Telangiectasia jejunum	1
Arteriovenous aneurysm jejunum	1
Carcinoma stomach	1
Total	31

TABLE 4

Lesions Found at Operation by Palpation	No. of Cases
Lymphosarcoma stomach	2
Leiomyoma stomach	1
Liposarcoma stomach	1
Carcinoid duodenum	1
Metastatic melanoma with erosion of duodenum	1
Carcinoma of pancreas with erosion of pancreatoduodenal artery	1
Pancreatic abscess with gastritis	1
Total	8

inspection has been the most productive of the methods herein described (table 3).

B. Palpation. Beginning at the cardioesophageal junction the fundus body and antral area of the stomach should be carefully palpated. Transmitted thrills may suggest the presence of aneurysm or tumors may be felt. Palpation of the portal triad through the foramen of Winslow must be carried out. Thrombosis of the portal vein or aneurysm of the hepatic artery may be noted. The duodenum and pancreas are also systematically examined. A goodly number of lesions are revealed by this exercise (table 4). If no information is gained by inspection or palpation, additional procedures are rapidly carried out.

C. Adjunctive Measures

1. *Transillumination.* If the operating room light is depressed to the level of the patient, examination of the mobilized duodenum (Kocher maneuver) and the proximal jejunum is facilitated. Intraluminal lesions will be outlined and enable a precise incision for investigation.[16] (table 5).

TABLE 5

Lesions Found at Operation by Adjunctive Measures	No. of Cases
Transillumination	0
Aspiration of gallbladder or common duct	
Pylephlebitis with erosion of hepatic artery	1
Acute cholecystitis with erosion of cystic artery	1
Portal pressures	
Portal thrombosis	1
Varices	1
Portagrams	
Varices	1
Cavernous transformation	1
Gastrotomy, duodenotomy, or endoscopy	
Peptic ulcer occulta	4
Varices	2
Mallory-Weiss syndrome	1
Total	13

2. *Aspiration of the gallbladder and the common duct.* Hemobilia is a source of massive hematemesis which is not generally considered. It may appear weeks after trauma so that the traumatic episode is not associated with the bleeding.[24] It also may arise from acute necrotizing infections of the gallbladder with erosion of the cystic or hepatic artery.[15, 28] A small needle is used to aspirate the gallbladder or the common duct, and the presence of blood will indict the liver or gallbladder as the source of hemorrhage (table 5).

3. *Portal pressures.* If dilated veins have been found on inspection, a secondary omental vein is mobilized, incised, and a polyethylene cannula is tied into it. Normal pressure in the portal system will usually be recorded at 80 to 180 mm. saline solution and pressures over this level should prompt a portagram (table 5).

3. *Portagrams.* If portal thrombosis is present or portal venous pressures are elevated, a portagram is performed. Hypaque (20 to 25 cc. of a 45 per cent solution) is injected rapidly through the cannula used for the measurement of portal pressure. The x-ray will nicely identify a block or cavernous transformation in the portal vein. Varices are often opacified by the dye. Most importantly if the bleeding from varices is due

to extrahepatic portal bed block, the portagram will reveal veins of adequate size (1 cm. or larger) for the performance of an emergency portacaval shunt (table 5).

5. *Adequate gastrostomy, duodenostomy and endoscopy.* These procedures have purposely been left to the last to avoid soiling the viscera with blood. An adequate gastrostomy is performed, and this is one that is large enough to permit inspection of all portions of the stomach with eversion of the stomach into the gastrostomy wound.[27] It should be large enough to admit the operator's hand. The lesser sac should be entered concomitantly so that palpation of the stomach is bimanual. After palpation a sterile lighted sigmoidoscope is introduced into the stomach through the gastrotomy wound. This enables direct inspection of the more inaccessible areas, *i.e.,* (cardio-esophageal junction lower ⅓ of the esophagus and the 1st and 2nd portions of the duodenum). If visualization of the duodenum is not satisfactory, it is opened and inspected and palpated directly (table 5).

6. *"Blind" gastrectomy.*[4, 19] If after these procedures, no identification of the bleeding lesion has been achieved, a decision must be made whether to back out or to perform a "blind" gastrectomy. This should be performed only as a last resort, and careful adherence to the outlined scheme will reduce the number of these gastrectomies. There has been guarded advocacy of this procedure for unfortunately, after its performance, re-bleeding has complicated the postoperative period. In this series there have been 7 patients in whom no source for the bleeding was found at operation. Five of these patients had so called "blind" gastrectomy performed, and 2 had bouts of re-bleeding in the postoperative period. Two patients had no definitive procedure carried out and 1 of these patients bled again (table 6).

The first consideration in the treatment of these patients is prompt resuscitation from the shock state and restoration of normal vital signs by administering a sufficient quantity of fresh whole blood. One specific physician must assume responsibility and carry out a definite plan. It is not possible to ascertain how much time one has for diagnostic studies, and it is, therefore, mandatory that only diagnostic procedures providing a maximum of information with a minimum of patient manipulation be done. It is possible to study these patients to death.

It is apparent that even with the use of the outlined methods for preoperative and operative phases of management our diagnostic accuracy preoperatively was 61.6 per cent in 32 patients. Of the 23 patients remaining undiagnosed before operation 16 of these or a further 16.7 per cent of the total were diagnosed at operation. The remaining 7 ((11.7 per cent) patients defied diagnosis at operation. In 5 patients a "blind" gastrectomy was carried out, and no information was provided by microscopic examination of the stomach and duodenum excised. Of these 5 patients 2 bled again in the postoperative period. One was controlled by vagotomy, and the other continued to bleed and died. At postmortem

TABLE 6

No Lesions Found at Operation*	No. of Cases
"Blind" gastrectomy	
No further bleeding	4
Rebleeding	2
Vagotomy with control	1
Deaths (postmortem revealed reticulum cell sarcoma of spleen with invasion of gastric fundus)	1
No procedure performed	
No further bleeding	1
Rebleeding, suspected malingerer	1

* No tissue diagnosis on stomach removed—8.

TABLE 7

Operative Mortality (1 to 30 days)	No. of Cases
Peptic ulcer (gastric and duodenal)	
Wound disruption (operative delay)	2
Operative deaths (irreversible shock)	2
Lower nephron nephrosis	1
Varices	
Hepatic coma	2
Hepatic coma and hepatoma	1
Rare lesions	
Reticulum cell sarcoma of the spleen with invasion of the gastric fundus (lesion missed at exploration, bled postoperatively)	1
Total	9

examination there was a reticulum cell sarcoma of the spleen invading the fundus of the stomach. Two patients in this group had no procedure performed, and one bled again. Malingering was suspected but not proven. Our operative mortality in these 60 cases was 15 per cent (table 7).

Our experience in this series prompts us to advise less delay in undertaking early operation. There is understandable reluctance to operate without a diagnosis, but it is evident that no amount of study would have revealed the diagnosis preoperatively in some of these cases presented. By an increasing adherence to this plan of preoperative and operative management we believe our operative mortality will improve and the postoperative bleeding and morbidity be lessened.

SUMMARY

1. Sixty cases of massive upper gastrointestinal tract bleeding requiring emergency operative intervention have been presented.

2. A plan for the preoperative and operative management of these patients is outlined.

3. Diagnostic accuracy preoperatively and during operation is discussed.

4. Rarer lesions causing massive hematemesis in this series are tabulated.

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TEN-YEAR EXPERIENCE WITH OPERATIVE CHOLANGIOGRAPHY

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INTRODUCTION

Operative cholangiography, since its introduction by Mirizzi[16] in 1932, has been widely accepted by many surgeons as a valuable adjunct to clinical impressions in order to evaluate the nature and extent of bile tract pathology. The clinical indications for common duct exploration have been well outlined by Colcock,[4] Glenn,[7] Traube,[19] and others. It is generally agreed that, on the basis of clinical indications alone, about 40 to 50 per cent of choledochostomies are unnecessary and lead to increased morbidity and mortality.

Operative cholangiography is now being thought of as a simple and safe method of reducing unproductive explorations of the common bile duct. The prevailing opinion is that this procedure is not the final answer to the complete abolishment of negative explorations, but represents one of the best solutions toward that end.

Today, from the existing literature, there appears to be two controversial aspects concerning this procedure. The first is whether operative cholangiography should ever be done; and, secondly, whether operative cholangiography should be employed on a selective or consecutive basis. The first aspect appears to be entirely a matter of personal preference.[3] The authors have followed both aspects of the second issue, namely selective and consecutive operative cholangiography. This report constitutes their experiences in series of cases which had both selective and consecutive operative cholangiograms.

METHODS AND MATERIAL

The technique of operative cholangiography employed by the authors has previously been described by Adams and Williams.[1] The technique consists of thorough exploration of the biliary system and the remaining abdominal contents after entrance into the peritoneal cavity. If, after careful inspection and palpation, the gall bladder is not needed for a bypass procedure, it is removed according to the techniques of the operating surgeon. A small (No. 5 to 6) ureteral catheter or polyethylene tube is then threaded into the common duct by way of the cystic duct stump for a distance of 2 to 3 cm. It is secured in place by a single tie of chromic catgut. The catheter is then cleared of any air bubbles by aspiration, and 5 to 10 cc. of 50 per cent Hypaque are injected slowly after removal of all instruments and sponges from the abdominal cavity and abdominal wall.

A 14 by 17-in. x-ray film in a 6:1 grid casette, which has previously been placed in a wooden tunnel beneath the patient, is exposed by using a 200-ma. portable x-ray unit. The machine is set at 75 to 80 kv. at 0.5 sec. at 40 in. distance.

Following exposure of the first film, a second film is placed and exposed in a like manner after the injection of an additional 10 to 15 cc. of 50 per cent Hypaque. Both films are developed simultaneously and then returned to the operating room for interpretation by the surgeon. While waiting for the films to be processed, the gallbladder bed is closed; and if time permits, the appendix may be removed at this point in the procedure.

From October 1949 through December 1959 a total of 989 cholecystectomies was performed by the authors, other staff surgeons and residents. From October 1949 to October 1953 operative cholangiograms were done on a selective basis, with the criteria of selection being entirely up to the surgeon performing the cholecystectomy. From October 1953 through December 1959 operative cholangiograms, before any decision concerning exploration of the common duct, were considered to be a routine part of the operative procedure except in certain instances where the additional time or the existing pathology was such that the procedure was considered not in the best interest of the patient.

During the entire period, post choledochostomy T-tube cholangiograms following common duct exploration were deemed a necessary and important part of the procedure as emphasized by Adams and Williams.[1]

RESULTS

Selective Group

During the 4-year period, when selectivity was practiced, there was a total of 408 cholecystectomies performed (table 1). Of the group 260 patients had no operative cholangiograms of any type; 97 patients had satisfactory cholangiograms before decision regarding exploration of the common duct; 17 patients had T-tube cholangiograms at operation only. There was a total of 34 patients whose films were considered unsatisfactory, or 26 per cent of the total patients having precholedochostomy cholangiograms attempted.

During the 408 cholecystectomies, 44 explorations of the common duct were performed. (table 2). Thirty-seven explorations were done on clinical indications alone; 19 explorations on the basis of clinical indications were positive while 18 explorations were negative. Five explorations were carried out on a basis of an abnormal cholangiogram; 4 of these were positive while 1 was negative. Two cases underwent common duct exploration on clinical indications but with cholangiograms interpreted as normal; both explorations were negative. By the use of clinical judgement, augmented by cholangiograms in about 25 per cent of the cases, 21 of 44 explorations (48 per cent) were negative.

Follow-up studies in this group have shown that in 7 cases pathology had been overlooked (table 3). At re-operation or autopsy, there were found 1 case of ampullary stricture, 5 cases with stones and 1 case with a large symptomatic cystic duct remnant.

Hospital deaths in this series total 10 (table 4) for a mortality of 2.4 per cent. Only 1 of the 10 patients had a common duct exploration, and 2 patients had an operative cholangiogram.

The only conclusions drawn at the end of this selective series were that, in the small series of cholangiograms done, the total number of explorations was reduced somewhat and that the procedure did not significantly contribute to an increased mortality. It was felt that selectivity did not reduce the number of negative explorations of the common duct except in that the total number of explorations was less.

It was felt that if the procedure was to have any value, in our hands, then it must be done without selection.

TABLE 1

Total cases—selective series

Total cases	408
No cholangiograms	260
Operative cholangiograms	97
T-tube cholangiograms only	17
Unsatisfactory cholangiograms	34

TABLE 2

Summary of exploration of common duct selective series

	No. of Cases	Positive Exploration	Negative Exploration
Cholangiogram suggesting pathology	5	4	1
Normal cholangiogram, positive clinical indication	2	0	2
No cholangiogram, positive clinical indication	37	19	18
Total	44	23	21

TABLE 3

Overlooked pathology—selective series

Type of Pathology	No. of Cases	Cholangiogram	Exploration of Duct
Common duct stones	5	Not done	2
Stricture	1	Not done	None
Cystic duct remnant	1	Not done	None

TABLE 4

Deaths—selective series

Cause or Underlying Pathology	No. of Cases	Cholangiogram	Exploration
Coronary thrombosis	2	2	0
Cerebral thrombosis	1	0	0
Cerebral embolus	1	0	0
Acute hemorrhagic pancreatitis	1	0	1
Pericarditis	1	0	0
Obstructive jaundice	2	0	0
Congestive heart failure	1	0	0
Cause not known, had simultaneous gastrectomy	1	0	0
Total	10	2	1

Consecutive Group

From October 1953 through December 1959 a total of 581 cholecystectomies was performed. During this time operative cholangiograms, before a decision concerning common duct exploration, were considered an essential part of the procedure in all cases except 46 (table 5). In these 46 patients, there were 7 explorations of the common duct, 6 cases in which the common duct stone was easily palpable, and 1 case to establish common duct drainage in a common duct which was totally blocked by massive tumor infiltration.

Operative cholangiograms were attempted in 535 cases as part of the procedure of cholecystectomy. In 62 cases the films were considered to be unsatisfactory for reasons listed in table 6, for a total of 11.5 per cent. In the group of patients who had unsatisfactory films, there were 4 explorations of common duct done on clinical indications alone. All 4 explorations were negative.

In the patients having satisfactory cholangiography, cholangiograms were interpreted as having indications of common duct pathology in 43 instances. In all 43 cases exploration was carried out (table 7). Stones were found in 25 cases, stricture in 6 cases, and exploration was negative in 9 cases. In 3 instances, the operator felt that he had caused passage of a stone through the ampulla by probe or dilator. During the consecutive series of 535 cholecystectomies, a total of 54 explorations (10 per cent) was done. Negative explorations totaled 24 per cent or one-half of the total negative explorations for the selective series.

During this time, there were 3 cases of proven overlooked common duct pathology (table 8). One case was the result of an unsatisfactory T-tube cholangiogram; 1 case was the result of misinterpretation of the T-tube cholangiogram; and 1 case of misinterpretation of operative cholangiogram. Two cases have required reoperation while the third is asymptomatic at present time without surgery.

Four cases had complications which possibly could be considered to have been caused by operative cholangiography (table 9). There were 2 cases of cholangitis, 1 case of acute hemorrhagic pancreatitis and 1 case in which perforation of the common duct was thought to have occurred. Exploration of the duct was negative for any

TABLE 5

Cholangiograms not done—consecutive series

Reason	No. of Cases
Palpable stone easily felt	6
Error in diagnosis	5
Inflammatory or tumor mass	14
Cholecystectomy done as part of other procedure	5
Poor condition of patient	1
Request of anesthesiologist (explosive gas; non-shock-proof machine)	1
No reason given	14
Total	46

TABLE 6

Unsatisfactory cholangiograms—consecutive series

Reason	No. of Cases
Unable to catheterize cystic duct	24
Catheter in duodenum	10
Failure of x-ray equipment	4
Barium in gastrointestinal tract	1
Extravasation of dye	5
Poor exposure, obesity, technical failure	6
No reason given	12
Total	62

TABLE 7

Summary of exploration of common duct—consecutive series

	No. of Cases	Positive Exploration		Negative Exploration
		Stone	Stricture	
Cholangiogram suggesting pathology	43	28	6	9
Unsatisfactory films, positive clinical indications	4	0	0	4
No cholangiograms	7	6	1*	0
Total	54	41		13

* Tumor.

recognized perforation. All patients with complications thought to be due to cholangiography have survived their complications.

Hospital deaths totaled 6 cases for an operative mortality of 1.1 per cent (table 10). Of the 6 deaths, 3 fall into the category listed in table 5 in that cholangiography was not done since it was thought that omission of the procedure served the best interests of the patient. Three cases had operative cholangiograms done. Only 1 of the 6 patients who died had exploration of the common duct.

TABLE 8

Overlooked pathology—consecutive series

Case	Pathology	Reason
1	Stone	Unsatisfactory T-tube cholangiogram
2	Stone	Misinterpretation of T-tube cholangiogram
3	Stone	Misinterpretation of operative cholangiogram

TABLE 9

Complications thought to be due to cholangiography—consecutive series

Complication	No. of Cases
Cholangitis	2
Pancreatitis	1
Possible perforation of common duct by catheter	1
Total	4

TABLE 10

Deaths—consecutive series

Cause or Underlying Pathology	No. of Cases	Cholangiogram Done	Exploration Done
Congestive failure	1	No	Yes
Congestive failure	1	Yes	No
Peritonitis with wound dehiscence	1	Yes	No
Concomitant gastrectomy with duodenal stump leakage	1	No	No
Metastatic carcinoma of liver	1	No	No
Cause unknown	1	Yes	No
Total	6	3	1

DISCUSSION

Partington, in a discussion of Hoerr's paper on operative cholangiography, states, "the most important thing about operative cholangiography, in my opinion, is its routine use." "... it is impossible to select the proper cases on which to do cholangiography."[13] This has been the opinion of one of us (J. M. E.) and consequently this practice of routine or consecutive cholangiography has been carried out for the past 6 years.

The decision for consecutive cholangiography was prompted by the results of our selective series. During this time there was a total of 44 explorations with 48 per cent being negative, plus the fact that a high failure rate regarding overlooked pathology was evident.

The high percentage of negative explorations and overlooked pathology was, in our opinion, due to two reasons: (1) that clinical indications alone for common duct exploration as presently accepted are not sufficiently accurate to reduce the number of negative explorations in our hands, and (2) experiences gained in the technique and interpretation of selective films were not sufficient to render the procedure as accurate as it might become.

It was felt that if operative cholangiography was to be of any benefit, then routine usage should be done whenever possible. Kantor and associates[14] and others believe that indications for operative cholangiography prior to exploration of the common duct should be at least the same as indications for common duct exploration. The authors of this paper do not share that view. In a series of 800 cholecystectomies, Hight and associates[12] found 20 cases of common duct stones by operative cholangiography when no clinical indications for exploration were present. We have had similar experiences.

Baker[2] states that dilation of the common duct should be a chief indication for either open exploration or operative cholangiography Traube[1] states that unless the duct is tremendously enlarged, its size can not be evaluated by inspection or palpation and recommends "a certain amount of dissection" over the anterior surface of the common duct. It is our feeling

that any dissection in this region is not without danger and that operative cholangiography gives an equally accurate appraisal of the size of the common duct.

In our series, there have been cases in which dilation of the common duct was readily apparent, but cholangiography has shown the biliary tree to be free of pathology. Follow-up in these cases, even at times with intravenous cholangiography, has proven that exploration of the common duct is not essential in the presence of dilation. Baker[2] suggests a possible explanation when he states that physiologic dilation of the common duct may result after cystic duct obstruction removes the gall bladder's function, just as it occurs after surgical cholecystectomy. Corff[5] in his series of 100 consecutive cases of common duct exploration, states that dilation was by far the most frequent indication for exploring the common duct, but was the least rewarding in the discovery of stones in the duct.

On the other hand, there have been patients in our series whose common ducts have been normal in size by palpation and by cholangiography and in whom common duct stones were found.

Jaundice can occur either directly or concomitantly with gall bladder disease without evidence of common duct pathology. Traube[19] states that only one-half of the patients who have gallstones and jaundice will be found to have stones in the common duct. Glenn[8] and others have observed stones in the common duct in patients who have only a large cholesteral stone. This should not be interpreted as advocating complete abandonment of consideration for clinical indications, but rather an abandonment of attempting to select which patients should have cholangiography. We do not agree entirely with Smoot and Cimmino[12] who feel that palpation of the duct for stones is fraught with so much uncertainty that it should be abandoned. It is agreed that palpation of the common duct is an uncertain procedure, however, even minimal palpation will occasionally lend itself to the discovery of stones and thereby render, in our opinion, a precholedochostomy cholangiogram unnecessary as indicated by 6 cases mentioned in table 5 in which cholangiogram was not done. It is this group, however, in which it is felt that postcholedochostomy T-tube cholangiogram at the time of operation is extremely important.

There is no hesitation in attempting catheterization of the cystic duct in the presence of inflammatory reaction unless such reaction has totally obliterated the cystic duct lumen or rendered the cystic duct so friable that handling is apt to produce laceration of the cystic duct in the immediate vicinity of the common duct. Attempting to tie off such lacerated cystic ducts may well lead to injury of the common duct. In the consecutive series, there were 49 cases of sub-acute cholecystitis and 99 cases of acute cholecystitis. In the 14 cases in which cholangiograms were not considered because of inflammatory or tumor involvement, 9 cases were due to extensive inflammation.

It is well established that some degree of jaundice can occur with acute cholecystitis.[9] The presence of common duct stones in Colcock[4] series was 24 per cent in cases of acute and subacute cholecystitis, or 3 times as frequently as in patients with chronic cholecystitis and cholelithiasis. Operative cholangiograms in the presence of acute or sub-acute cholecystitis with jaundice are extremely helpful in making a decision as to whether exploration of the common duct is necessary. There appears to be no increased incident of overall complications following catheterization in the presence of inflammatory changes.

The 62 cases which had unsatisfactory films represents 11.5 per cent of the total precholedochostomy cholangiograms taken. This compares favorably with other reports by Mixter and associates,[17] 7 per cent; Hight and Lingley,[11] 16 per cent; Douglas,[6] 33 per cent; and Kantor,[14] 48 per cent. Of the films 11 per cent were unsatisfactory and represents what we feel to be more than an acceptable average. The use of a 200-ma. x-ray machine instead of the less powerful 12-ma. machine has caused the quality of the x-ray to improve tremendously. However, as seen from table 6, errors in technique account for the majority of unsatisfactory films. Routine usage of cholangiography represents the best method of reducing such unsatisfactory films by allowing the surgeon to develop and practice his own techniques. Lack of experience in the procedure will always be a factor in producing unsatisfactory films until surgeons develop familiarity with the techniques involved. Of course, unsatisfactory films are not limited entirely to the inexperienced surgeon, and even the

most experienced surgeons will produce faulty films at times.

In this series, refinement of technique may well have prevented the 4 unnecessary explorations which occurred in cases with unsatisfactory films.

The 9 cases with false positive interpretations and subsequent negative explorations represent an area for improvement. Five of the negative explorations occurred during the first 6 months and 7 in the first year, while over the next 5 years only 2 negative explorations were done on patients with interpretable cholangiograms. Routine usage of the procedure has, in our opinion, played a very definite part in this reduction of negative explorations. For the past 5 years, our procedure has been altered so that during this time two films instead of one are obtained following cystic duct catheterization. This change in technique has also helped reduce the negative explorations, by allowing a better interpretation of films suspected of having air bubbles instead of stones in the extra hepatic biliary tree.

Our procedure calls for the films to be developed on the operating floor and to be read in the operating room by the surgeon performing the procedure. We feel that surgeons can and should develop the ability to interpret their own films, with undecided situations being discussed by both surgeon and radiologist.

The authors feel that it is unwise and unfair to ask the radiologist to give so definite an opinion on a case he has never seen or for the surgeon to accept an opinion without seeing the films whose creation has largely been through his efforts. Discussions over difficult interpretations which arise are best settled in the operating room with both radiologist and surgeons participating.

The 3 cases of overlooked pathology in the consecutive series represents 0.5 per cent of all cholecystectomies and 5 per cent of cases undergoing common duct explorations. Our period of follow-up is not sufficiently long to render these figures valid.

While operative cholangiograms have as their main function the detection of common duct pathology before exploration, we feel that it serves other very definite purposes. The next most important function is, of course, detection of overlooked pathology after exploration of the common duct (fig. 1). Mixter and associates[17] report a 13 per cent incidence of overlooked stones that were found by T-tube cholangiogram done at the operating table before closure of the abdomen.

FIG. 1. Large residual common duct stone demonstrated by T-tube cholangiography after exploration of common duct.

FIG. 2. Cystic duct stump (2.5 cm.) demonstrated by operative cholangiography. Stump re-amputated 5 mm. from common duct junction after further dissection.

During the past 6 years, there have been instances in which cholangiography has demon-

FIG. 3A. Small common duct inadvertently divided during cholecystectomy. Identification of distal portion of divided duct by cholangiography.

FIG. 3B. Identification of proximal portion of divided common duct

FIG. 3C. Demonstration of patency and security of anastamosis of divided common duct by T-tube cholangiography.

strated long cystic duct remnants (fig. 2). Such remnants[8, 15] can then be further dissected and re-amputated .3 to .5 cm. proximal to its junction with the common duct at the original procedure and thereby save a possible re-operation for symptomatic remnants. Two such cases of symptomatic remnants have been done in the past several years, neither of which had cholangiography at the time of the original cholecystectomy.

Cholangiography has been used successfully to demonstrate the patency and security of choledocoduodenostomy after two instances in which the common duct was damaged during gastrectomy. Although the occasions for this type of usage are fortunately rare, the demonstration by cholangiography of a leaking anastomosis and its subsequent repair may well lead to a reduced incidence of biliary fistula.

In 1 case (fig. 3), cholangiography was used for identification of an extremely small common duct after it had been inadvertently divided during cholecystectomy. The patency and security of anastomosis was also demonstrated. This patient did develop a transient cholangitis which is asymptomatic now 2 years since his operation. Although we have had no opportunity to use cholangiography in operations for obstructive jaundice in the newborn, we feel that it would be of definite benefit as emphasized by Gross.[10]

The operative deaths in the selective series was 2.4 per cent and in the consecutive series 1.1 per cent. In no case do we feel that the additional time and the injection of radiopaque material into the extra hepatic biliary system has been responsible for any patient's death.

CONCLUSIONS

Although the technique and results of operative cholangiography have still room for improvement, it is felt that the procedure is definitely worth the additional time and effort to achieve the desired results. Operative cholangiography has been shown to reduce the number of explorations of the common duct. By the use of the technique in consecutive cholecystectomies, the incidence of negative explorations and overlooked pathology has been reduced to one-half as compared to selective usage. There has been no increase in the mortality due to cholangiography on a consecutive basis.

The authors recommend consecutive or routine

employment of operative cholangiography in all cases undergoing cholecystectomy whenever possible, except in certain cases when a precholedochostomy operative cholangiogram is not indicated because a common duct stone is easily palpable. Emphasis should be placed on obtaining satisfactory postcholedochostomy operative T-tube cholangiograms in these cases.

SUMMARY

1. A ten-year experience with selective and consecutive cholangiography has been presented.

2. Results in selective and consecutive series have been compared.

3. The value of cholangiograms in various phases of extrahepatic biliary tract surgery has been presented.

4. Conclusions are drawn regarding the authors' opinions concerning the procedure of operative cholangiography.

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THE SURGICAL APPROACH TO INTRATHORACIC GOITER

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Much misunderstanding exists in the medical profession today concerning intrathoracic goiter. The term "substernal goiter" is commonly used by us all, yet the question of the origin of mediastinal thyroid masses, their classification, and most important of all, how these lesions should be surgically approached for safe removal, is as controversial a subject as one would care to find. Lack of understanding of certain of these factors led me into an untenable position at the operating table a few years ago and cost the life of the patient. I learned a very important lesson the hard way and have more recently operated upon a patient with a similar problem quite safely and successfully as a result of a newer understanding. I feel that perhaps this important lesson is not fully appreciated by all surgeons handling these problems, and it is for this reason that I am presenting this subject today.

CLASSIFICATION

By definition, the term "intrathoracic goiter" refers to a goiter the largest part of which lies below the superior thoracic aperture. Classification of these goiters is rather cumbersome. Such terms as substernal, posterior mediastinal, plunging, aberrant mediastinal, total intrathoracic, partial intrathoracic and others make accurate classification difficult. According to time-honored texts of anatomy, the mediastinum is composed of a superior and an inferior portion, the division being made by an imaginary line passing from the 4th thoracic vertebral interspace to the junction of the manubrium with the body of the sternum. This line passes through the carina. The inferior mediastinum is divided into anterior, middle and posterior compartments, the heart occupying the middle. The superior mediastinum, where most intrathoracic goiters are found, contains no accepted subdivisions. From a practical standpoint, however, it is most useful to consider the superior mediastinum to consist of an anterior or substernal portion, and a posterior portion, with the trachea separating the two. Thus intrathoracic goiters shall almost always be either substernal or posterior mediastinal. Granted in the latter case the mass may occupy the posterior portion of the superior mediastinum or the true posterior mediastinum or both, but from the surgeon's standpoint these may well be considered as one. Most of the larger posterior mediastinal goiters which have been reported extend into the posterior aspect of both superior and inferior mediastina.

ORIGIN

The origin of these intrathoracic masses is an unsettled question. Embryologically, the thyroid gland originates from a median thyroid component arising as a single median pharyngeal derivative, and 2 lateral thyroid components from the lateral walls of the 4th pharyngeal pouches. The parathyroid glands originate in the 3rd and 4th pouches, and the thymus, although details are debatable, seems to arise in the lateral pharynx adjoining the lateral thyroid components.[12] Thus the close proximity of the early stages of development of these various glands to some extent explains the possibility of aberrant positions in their anatomical relationships. All of us are aware of the fact that parathyroid adenomas may be found in the mediastinum, usually in close proximity to the thymus gland. Based on similar reasoning, cases of intrathoracic goiter have been said to originate as aberrant thyroid tissue especially if not in direct continuity with the cervical thyroid. Many students of the subject, however, prefer to consider them as cervical thyroid. Many students of the subject, however, prefer to consider them as cervical goiters which have gradually descended into the chest. I make no claim to settle the dispute.

The observed fact that cervical goiters tend to descend into the chest is probably best explained by the fact that the downward direction represents the path of least resistance to an enlarging goiter with relatively tight musculofascial structures lying anteriorly and laterally, and the esophagus with underlying vertebral column posteriorly. The pretracheal layer of the deep

cervical fascia which encompasses the thyroid gland extends directly downward into the mediastinum to eventually become fused with the fibrous pericardium, thus providing a natural pathway for enlarging masses to follow. Also a downward sucking effect caused by the negative intrathoracic pressure with each inspiration is probably a 3rd contributing factor.

The question of *blood supply* to these intrathoracic goiters is of paramount interest to the surgeon, for control of this blood supply is essential for safe excisional surgery. The most frequently encountered mediastinal goiter is the *substernal* type which usually results from the downward growth of a cervical goiter from the lower pole or isthmus of the gland. These goiters lie in the superior mediastinum, anterior to the major vessels of the neck and mediastinum. The blood supply to these substernal goiters is almost always from the inferior thyroid arteries and, perhaps, the thyroid ima. Not infrequently, however, significant blood supply enters from the various mediastinal vessels, both arteries and veins, such as the aortic arch, innominate, internal mammary, and vena cava.

The *posterior mediastinal* goiter, on the other hand, seems to arise from the posterior and lateral aspect of the lateral lobe (rather than the inferior pole)[19] and descends posterior to the carotid sheath and recurrent nerve, and posterior to all the great vessels of the superior mediastinum, coming to rest against the vertebral column. These descending masses are deflected by the aortic arch and the large majority extend into the right chest rather than the left, even though the origin may be from either lateral thyroid lobe. Most carry with them a relatively narrow stalk of thyroid tissue containing thyroid vessels maintaining continuity with the cervical thyroid, although some seem devoid of any cervical connection. Many of these posterior mediastinal goiters are found to have sizeable vascular supply from the various mediastinal vessels regardless of whether or not a cervical stalk is present. A number of cases have been described including one of the cases herein reported in which a plexus of quite large veins covers the goiter,[15] making it resemble a massive varicocele. Most of the above anatomical details have been clearly described by Sweet.[19] The prime surgical significance of the above seems to me to be the fact that many or most of these large intrathoracic goiters have *both a cervical and a mediastinal blood supply*, and when a surgical approach is planned, it must provide for control of all contributing vessels.

INCIDENCE

It is interesting to note that the reported incidence of posterior mediastinal goiter has increased considerably since the field of thoracic surgery has come of age. Mora[16] in 1944 could find only 6 cases of posterior mediastinal goiter reported and added 1 of his own. Sweet[19] reported 6 such cases in 1949. Ellis[5] in 1952 reviewed the intrathoracic goiters removed by other routes than the cervical incision at the Mayo Clinic and found that of 24 such cases, 10 were substernal and 14 posterior mediastinal. Of the 24 cases 18 presented on the right side. This report did not, however, take into account the many substernal goiters removed by the classical surgical approach. Johnston[9] in an excellent discussion of this subject in 1956 reported 14 intrathoracic goiters of which 4 were substernal, 9 posterior mediastinal, and 1 rested on the diaphragm. Of 31 intrathoracic goiters recorded by Lindskog[11] in 1957, 69 per cent were substernal and 31 per cent posterior mediastinal. A number of other reports have appeared in the recent literature.[2, 4, 6, 10, 12-15, 17, 20] A study of this subject matter forces the conclusion that posterior mediastinal goiter is not nearly so rare as was thought a few years ago.

DIAGNOSIS

Quite frequently intrathoracic goiter is completely asymptomatic, but when symptoms do appear they are primarily due to pressure on surrounding structures. Various of the following symptoms may be produced: dyspnea, cough, a sensation of fullness, mild pain, wheezing, dysphagia, stridor, hoarseness, a choking sensation, and hemoptysis. Dilation of superficial veins is seen with some of the larger goiters, and thyrotoxicity has occasionally been reported. From a diagnostic standpoint these masses must be differentiated from the group of other mediastinal tumors and cysts. The presence of a cervical goiter or a history of thyroidectomy is of considerable significance in the differential diagnosis.

McCort[15] reviewed the records of the Massachusetts General Hospital and found 28 cases of

mediastinal goiter. A study of these yielded the following significant roentgen findings:

(1) displacement of the trachea, in 27 of the 28 cases; (2) displacement of the trachea beginning high in the neck, frequently at the larynx; (3) compression of the trachea often present but not marked in degree; (4) displacement or compression of the esophagus; (5) upward motion of the goiter on swallowing, observed in 84 per cent of cases; (6) calcification within the mass, noted in 25 per cent; (7) a smooth or only slightly nodular outline of the tumor; and (8) reflection of the mediastinal pleura below the goiter.

A substernal goiter commonly deviates the trachea laterally in either direction and perhaps posteriorly. The posterior mediastinal goiter, on the other hand, produces rather striking anterior tracheal displacement as viewed on a lateral chest film in addition to lateral deviation on a postero-anterior film. These masses have no expansile pulsation, but at times angiograms are necessary for exclusion of the possibility of aneurysm. Falor[6] described atelectasis of the right lower and middle lobes produced by pressure of a mediastinal goiter and relieved by bronchoscopy. Case 1 exhibited complete atelectasis of the entire right lung from compression of the main stem bronchus by a massive posterior mediastinal thyroid. Such complications, however, are infrequently encountered.

Uptake studies of radioactive iodine have been used by a number of authors[4, 6, 8, 10] and may be quite helpful in diagnosis provided the structure of the goiter is functioning sufficiently to concentrate iodine. This is not always the case, however.

Microscopic study of removed intrathoracic goiters usually reveals them to be adenomatous thyroid of the colloid or fetal type. Carcinoma is rarely found.

SURGICAL ASPECTS

Once the diagnosis of mediastinal thyroid is made or strongly suspected, it is generally held that the tumor should be removed regardless of the presence or absence of symptoms. This seems sound advice for the large majority of such goiters show continued growth, and as a rule, the larger the tumor, the more difficult its removal.

The question of the surgical approach to these tumors is all-important. First, I should like to state and strongly emphasize that the vast majority of cervical goiters with lesser degrees of mediastinal extension can and should be removed through the usual collar type *cervical incision.* Thus it is only the exceptional case which requires additional surgical exposure. Many highly competent surgeons still prefer to use the "morcellation" method, popularized by Dr. Frank Lahey, in which the tumor is bluntly broken up into small bits and thus blindly removed. Unquestionably this technique has been successfully used many times, but it appears to me that in the present day era of safe thoracic surgery, direct visualization of the tumor, accurate ligation of its varying blood supply and *in toto* removal is both a more esthetic and a safer operation. It is my opinion that the large substernal thyroid which cannot be delivered into the neck without morcellation should be approached by a *median sternotomy* as an extension of the cervical incision. With the use of appropriate sternal shears, this is neither a difficult nor dangerous feat and allows clear visualization of the mass to be removed with accurate control of its blood supply.

It is the problem of the posterior mediastinal goiter, however, which I should most like to emphasize. These large tumors defy removal by a cervical incision alone. Most authors have stated a preference for a posterior thoracic incision for the necessary intrathoracic manipulation and excision of this portion of the tumor. If this approach is used, the patient must then be repositioned for cervical thyroidectomy or the operation must be performed in two stages as the majority are in direct continuity with a cervical goiter. This has obvious disadvantages, and from a limited but quite vivid personal experience, I can assure you that it is not always a simple task to control bleeding from the cut surface of the thyroid stalk extending up into the neck. This tissue is friable, holds sutures poorly, and serious hemorrhage may occur despite one's best efforts at control. I am firmly convinced that the safest method for removal of the posterior mediastinal goiter is the concomitant use of the classical *cervical incision plus an anterior intercostal incision,* usually the second, on the appropriate side. Costal cartilages above and below this incision may be divided with ease, and quite adequate exposure of the mediastinal mass is accomplished. Thus, the cervical and mediastinal blood supplies are controlled

under direct vision, and the entire mass can be removed, frequently without dividing the connecting stalk which is usually present. The recurrent laryngeal nerves and the major mediastinal vessels are visualized and protected.

This combined approach was described by Hart[7] in 1950 and has been successfully used by Maurer[14] and also by Johnston[9]. It is apparent, however, that this technique has not been widely recognized or utilized. The 2 cases I shall document have firmly convinced me of the soundness of the principle involved—direct control of the blood supply both from above and from below.

Should there be any doubt in the mind of the operator concerning the feasibility of removing the intrathoracic goiter by means of a cervical incision alone, he should in preparing and draping the patient include the neck, sternum, and anterior chest so that if the need becomes apparent, either a median sternotomy or an anterior thoracotomy may be carried out to allow adequate exposure for safe removal of the entire tumor.

CASE REPORTS

Case 1. G. G., a 36-year-old Negro woman was first seen in December 1951 at which time she was 7 months pregnant. A routine chest x-ray taken shortly before had shown the presence of a large posterior mediastinal tumor presenting on the right side. She was asymptomatic at that time as far as the chest lesion was concerned, and the decision was made that she should be allowed to deliver, following which the mediastinal tumor would be removed. During the subsequent month, symptoms of increasing shortness of breath followed by hemoptysis appeared, and she was admitted to the hospital on January 15, 1952. At that time she was in acute respiratory distress with marked shortness of breath and hemoptysis. She was unable to lie down and spent the night sitting on the side of her bed leaning forward over a table in order to get her breath. She was cyanotic and exhibited rather marked dilation of superficial veins over the upper chest, especially on the right. She gave a history of having had a thyroidectomy in 1949. Examination of the neck showed the presence of a large recurrent cervical goiter measuring 12 by 8 by 6 cm. on the right side. X-rays of the chest showed "complete obliteration of the right hemithorax due to either massive pleural effusion or a large tumor mass completely filling the right chest. The left lung field is clear. The cardiac shadow cannot be evaluated due to obliteration of the right side of the chest." Unfortunately, films of the chest taken at this time have been discarded and are not available for demonstration today. The film which is shown (fig. 1) was taken some 3½ years before operation. Needless to say, the tumor had shown massive growth since that time, sufficient to produce complete atelectasis of the right lung.

It was fully realized that this patient presented a desperate problem and it seemed obvious that surgical relief was necessary for her survival. The right chest was entered on January 19, 1952, through the bed of the subperiosteally resected right 5th rib posterolaterally. The diaphragm was extremely high and presented in the incision. The entire right lung was completely atelectatic and was hardly larger than a man's fist. The remainder of the right chest was filled with a large rounded tumor mass presenting from the posterior mediastinum and extending well below the azygos vein. The mass lay posterior to the trachea and the major vessels including the superior vena cava. The visible portion of the tumor was covered by a formidable plexus of tremendously large veins some of which were the size of a man's finger. This gave the appearance of a large varicocele. The tumor produced atelectasis of the right lung from occlusion of the right stem bronchus; there was also some compression of the trachea. An attempt was made to free the tumor from its capsule by ligating and dividing the massive veins. This was a rather bloody procedure but was successful for about ¾ of the distance around the tumor,

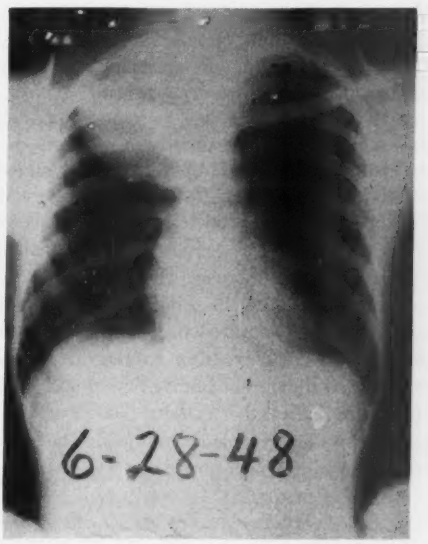

FIGURE 1

after which bleeding became profuse. The mass was removed as rapidly as possible except that it could not be freed at its upper extremity, and there was found a stalk of thyroid tissue extending through the superior thoracic aperture, apparently continuous with the goiter in the neck. It then became apparent that the entire lesion could not be removed through this incision, and pedicle clamps were placed across the upper margin of the tumor stalk in the posterior mediastinum. The clamps tended to cut through, the stalk held sutures very poorly, and a great deal of blood was lost before the wound was made dry. Up to this point, 2½ hr. of operating time had elapsed, and the patient had received about 7 pints of blood which we thought was fairly adequate replacement. After the tumor was removed, the right lung expanded without difficulty and the patient's systolic pressure was 100 mm. Hg. The usual anterior and posterior catheter drains were inserted and closure of the wound was begun. After the first row of sutures was placed, it was noted that the patient had no pulse, blood pressure, or respiration. The incision was rapidly reopened, and the heart was found to be in a state of standstill. Cardiac massage was carried out but return of the heart beat was not accomplished. Immediate Caesarean section was performed, and although the baby breathed a few times, he did not survive.

The specimen removed measured 8 in. in diameter and on microscopic study was found to be a massive encapsulated fetal adenoma of the thyroid.

Comment. I believe that 2 major mistakes were made in the handling of this case. The first was failure to advise operation when the patient was asymptomatic and 7 months pregnant. Within 1 month the right lung had become completely atelectatic, and the patient was cyanotic and markedly dyspneic, making operation much more hazardous. The 2nd mistake which was made was the selection of a posterior thoracic incision rather than a cervical incision combined with an anterior thoracic incision. Had this combined approach been used, bleeding could have been much more easily controlled. It is my impression that the terminal event of cardiac arrest was precipitated by chronic anoxia due to atelectasis of the lung, plus rather massive blood loss which perhaps was not completely replaced. The added burden of an 8-month pregnancy was possibly a contributing factor.

Case 2. Mrs. C. J., a 57-year-old white woman was admitted to the hospital on September 19, 1957, complaining of choking and shortness of breath. Five years before admission, she had been told that she had a substernal goiter. She was asymptomatic until the past few months when she began experiencing mild choking sensations on lying down. About 1 hr. before admission she had acute onset of choking, wheezing, and great anxiety. Examination of the patient by Dr. C. Warren Irvin revealed her to be in acute distress with wheezing apparently due to tracheal obstruction. She was quite frightened but coughed up a plug of mucous and then was able to breathe a great deal easier. General physical examination was otherwise not remarkable except for a sensation of fullness in the right side of the neck in the region of the thyroid gland. No distinct mass, however, could be felt. X-rays of the chest (figs. 2 and 3) showed a rather large mass in the posterior mediastinum presenting on the right side. Protein-bound iodine (PBI) was 9.8 mg. per cent. A diagnosis of posterior mediastinal goiter was made, and on September 24, 1957, the patient was operated upon utilizing a right anterior 2nd interspace incision with division of the 2nd and 3rd costal cartilages. The mass was found to be fairly typical thyroid tissue, presenting in the posterior medi-

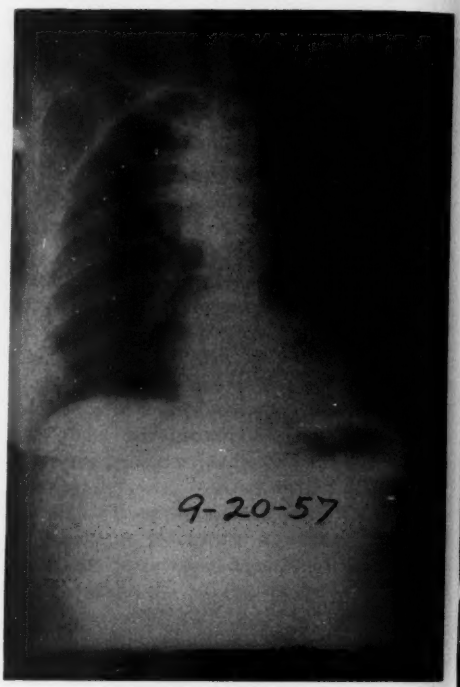

FIGURE 2

FIGURE 3

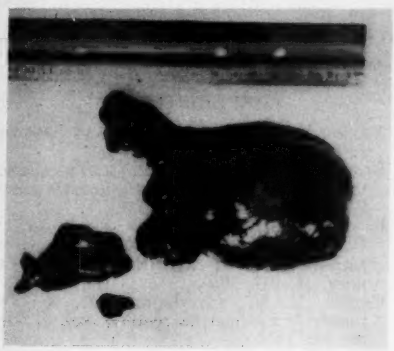

FIGURE 4

astinum posterior to the superior vena cava and trachea and lying on the vertebral column. It extended down to the azygos vein. It was rounded but was not covered by a plexus of veins as seen in case 1. The tumor was rather easily dissected out of the mediastinum but was found to have a broad pedicle which extended upward into the neck posterior to the great vessels and trachea.

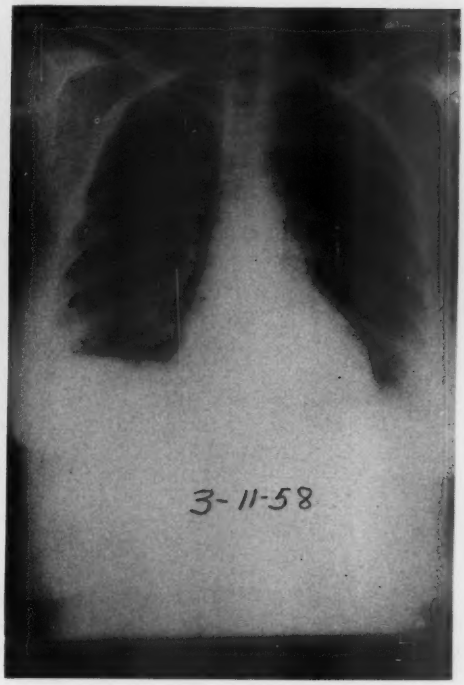

FIGURE 5

There was rather marked narrowing of the trachea, and dissection in the mediastinum at times almost completely occluded the airway so that dissection could be carried out only intermittently. A transverse cervical collar incision was then made and the thyroid gland approached in the usual manner. There was marked venous engorgement of the veins in the region of the thyroid. The right lobe was slightly enlarged and nodular, and there were 2 small thyroid nodules completely separate from the main thyroid body but adjacent to it. Cursory examination of the left thyroid showed it to be only slightly thickened, and it was my impression that the mediastinal goiter probably arose from the right side. Accordingly, virtually total right thyroidectomy was carried out, and it was then realized that the mediastinal goiter communicated with the left lobe of the thyroid rather than the right. The left lobe was freed and was retracted into the chest through the mediastinum, and the entire mediastinal goiter plus the left lobe were removed *in toto*. A small cigarette drain was left in the mediastinum and brought out the cervical incision. This wound was then closed in the usual manner. A posterior

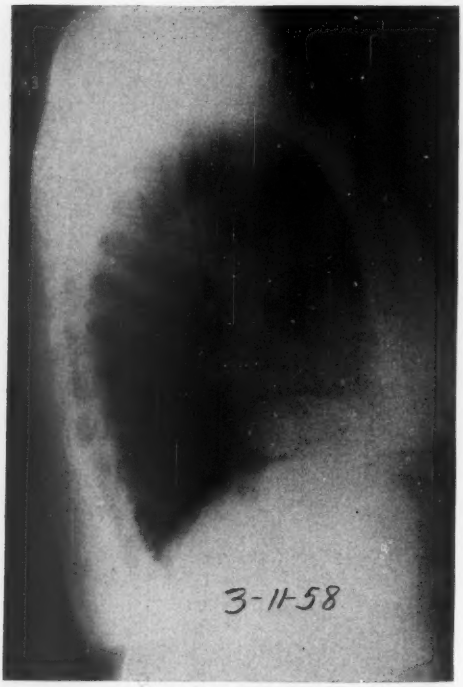

FIGURE 6

catheter drain was inserted into the thoracic cavity, and this wound was then closed. The patient received 4 pints of blood and tolerated operation well. Postoperatively, there was temporary difficulty in evacuation of tracheobronchial secretions, but this was no major problem, and the patient was discharged from the hospital on the 11th postoperative day. The mediastinal goiter measured 12 by 8 by 6 cm. and microscopically showed nodular hyperplasia (fig. 4). The patient's symptoms have been completely relieved, and she is well to date (figs. 5 and 6).

Comment. This patient had a typical posterior mediastinal goiter which was safely removed with a fairly large bilateral cervical goiter, utilizing the combined cervical and anterior thoracic approach. In retrospect, a tracheotomy probably should have been performed at the completion of the procedure.

SUMMARY

1. "Intrathoracic goiter" refers to any goiter the largest portion of which lies below the superior thoracic aperture. Most such tumors are either substernal or posterior mediastinal.

2. The embryology of the thyroid gland and its possible relationship to the origin of these goiters is discussed.

3. Most intrathoracic goiters have both a cervical and a mediastinal blood supply.

4. Symptomatology is related to pressure upon surrounding structures. The trachea is deviated, characteristically quite high in the neck, and the mass can usually be seen to move upward on swallowing. Calcification is noted in about 25 per cent of cases.

5. The large majority of substernal goiters can be safely removed by means of the standard cervical thyroid incision.

6. The unusually large substernal goiter which cannot be delivered into the cervical incision *in toto* is best removed by means of a median sternotomy.

7. Most large posterior mediastinal goiters are most safely removed utilizing the combined cervical and anterior thoracic intercostal approach.

8. Two cases of posterior mediastinal goiter are reported, one with poorly planned surgery and death of the patient, and the other operated upon utilizing the combined cervical and anterior thoracic incision with a gratifying result.

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THE MIMICRY OF TUMORS OF THE SPINAL CORD*

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The importance of early and accurate diagnosis in the treatment of disease is vital, for without it, our efforts are reduced to the uncertainties and the hit-and-miss methods of empiricism. To be sure, errors or delay in the diagnosis of some diseases may not lead to untoward results, but in others the end result of such errors or delay may prove to be unsatisfactory, if not disastrous. In the latter group, tumors involving the spinal cord must be considered high on the list. Tumors arising from the substance of the cord itself, intramedullary tumors, rarely respond satisfactorily to treatment. Fortunately, however, benign tumors within the spinal canal, which originate from the arachnoid and dural envelopes surrounding the cord and from the sheaths of the nerve roots—the meningiomata and neurofibromata (neurilemmoma, perineurial fibroblastoma, and schwannoma)—are more common.

SYMPTOMS

In the early stages of their development, many of these tumors produce the same symptoms as some other diseases, thus leading to improper treatment, including unnecessary surgical operations. Almost without exception, patients with tumors of the spinal cord have been treated for numerous nonexistent or irrelevant disorders before the correct diagnosis has been made. When confronted with the problem of pain in the back, the trunk, or the extremities, it is quite natural and usually correct for the physician to consider primarily the likelihood of the more common conditions, such as orthopedic disorders or diseases of the thoracic or abdominal viscera. Although the incidence of tumors within the spinal canal is comparatively low, the possibility of such lesions must be borne in mind if harmful delay and unnecessary disability are to be avoided.

Pain is often the initial symptom of an extramedullary tumor, because of the tumor's proximity to a sensory nerve root. Such pain may be localized to the spine, or it may be referred to the part of the body supplied by the involved nerve root. Thus it may simulate angina pectoris; biliary or renal colic; or the pain of pleurisy, pelvic disease, arthritis, neuritis, myositis, sacroiliac disorders, protruded intervertebral disk, coccygodynia, and other painful conditions. Continued growth of an extramedullary tumor within the rigid, bony confines of the spinal canal eventually results in compression of the spinal cord, with the development of numbness and motor weakness below the level of compression and sometimes with dysfunction of the sphincters of the bladder and of the anus. Disorders of gait, spasticity, hyperactive tendon reflexes, ankle clonus, and Babinski's sign may ensue. At this stage of its evolution, the neurologic nature of the disease becomes obvious, and there is little chance of mistaking it for a lesion outside of the nervous system. This clinical picture, however, may be mistaken for another nonneoplastic condition of the spinal cord, such as multiple sclerosis, amyotrophic lateral sclerosis, posterolateral sclerosis of pernicious anemia, myelitis, circumscribed arachnoiditis, epidural abscess, or syphilis of the cord.

If spinal neoplasm is not suspected and treated, continued compression may lead to serious and irreversible changes in the structure of the spinal cord. Prolonged severe compression is not well tolerated, although gradual compression, as by a benign extramedullary tumor, is often accompanied by neurologic changes that may not be permanent and that may be greatly improved by the release of pressure after the tumor is removed. Sudden compression, such as that caused by trauma or by metastasis of a malignant lesion to the vertebrae, is more likely to result in irreversible damage to the cord and consequent invalidism or death.

Almost all patients who eventually are found to have spinal cord tumors have received one or more of the following forms of therapy before establishment of the correct diagnosis: osteopathic or chiropractic manipulations, various

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types of injections and nerve blocks for relief of pain, injections of sera and vaccines, physical therapy (diathermy and massage), roentgen therapy, treatment for arthritis or sciatica, application of braces and belts, and a variety of surgical operations (spinal fusion, fasciotomy, abdominal exploration and removal of various viscera, nerve resection, rib resection, sacroiliac fusion, and nephropexy).

DIAGNOSIS

Diagnosis of spinal cord tumor in its early stages, before the development of the irreversible changes caused by pressure, depends primarily upon *bearing in mind the possibility of such a lesion.* The referred pain may be sharp and lancinating, with radiation to arms, legs, thorax, or abdomen, and may be initiated or aggravated by coughing, sneezing, or straining at stool. The pain is often worse at night and may prevent sleep. Neurologic examination may demonstrate a "sensory level" below the site of the tumor and motor and reflex changes of varying degrees. When the level of a tumor can be fairly well identified by symptoms and signs, the spine should be examined roentgenographically, centering upon the suspected site of the lesion. The films are carefully studied for evidence of any erosion of the pedicles or vertebral bodies, and for any increase in the width of the spinal canal or of the intervertebral foramina. The most important aid in the diagnosis of spinal cord tumors is lumbar puncture, including the Queckenstedt test and chemical examination of the cerebrospinal fluid for total protein content. With the lumbar puncture needle in the subarachnoid space and an attached glass manometer in place, the Queckenstedt test is performed by manually compressing the jugular veins in the neck. The presence of a tumor within the spinal canal above the site of the needle almost invariably prevents the column of cerebrospinal fluid from rising in the manometer during compression of the jugular veins. This spinal subarachnoid block may be complete or partial. When the block is incomplete, the rise of the column of fluid is slow, and when the jugular compression is released, the fall is gradual and may not reach the former level. Chemical examination of the sample of fluid removed below the level of a tumor commonly discloses an elevated total protein content (more than 45 mg. per 100 ml.). Not uncommonly the total protein is as high as 100 to 300 mg. per 100 ml., and in the presence of tumors of the conus medullaris or of the cauda equina the quantity may be as great as 1,000 to 3,000 mg. per 100 ml.

When the neurologic examination and the routine roentgenographic examination of the spine fail to identify the exact location of an intraspinal tumor, it is often necessary to resort to contrast myelography with Pantopaque. Usually 2 or 3 ml. of the radiopaque medium is sufficient to enable location of a tumor. The material may be injected into the lumbar subarachnoid space or into the cisterna magna. This procedure is carried out with the patient lying on a fluoroscopic table that can be tilted to permit observation of the movement of the opaque medium in the spinal canal toward the patient's head and feet. The tumor blocks or delays the flow of Pantopaque, and spot films are helpful in locating the tumor exactly.

TREATMENT

As soon as the diagnosis and the location of the spinal cord tumor have been established, laminectomy should be performed and the tumor removed, if possible. The benign neurofibromata and meningiomata, as pointed out earlier, can usually be totally removed, thus releasing disabling compression of the cord. Prompt diagnosis and operation may mean the difference between permanent invalidism with paraplegia, and relief of pain with restoration of neurologic function. The prognosis for tumors that are found to be within the substance of the spinal cord is, with few exceptions, unfavorable.

CASE REPORTS

In order to emphasize the importance of early diagnosis, I should like to summarize briefly several illustrative cases.

Case 1. In 1943 an adult man was examined in the Department of Orthopedic Surgery of the Cleveland Clinic because of increasing cramp-like pains in the muscles of both legs during a 7-year period. Soon after the onset, a lumbosacral fusion was performed elsewhere; it did not bring about relief. The pain forced the patient to give up his work as a barber. The pain was increased by flexion of the trunk at the waist, and the patient wore a brace. In our Department of Orthopedic Surgery a diagnosis of myositis was made, and typhoid fever therapy, local application of heat, and colchicine were recommended; lumbar puncture was advised,

but was refused by the patient. Two years later the patient returned to the Orthopedic Department here because the cramping pain in both legs continued. He was at once referred for consultation with a neurosurgeon. It was learned that his pain had been almost continuous for 10 years, and was worse on coughing and sneezing. There was no history of injury or of sphincter disorder. The neurologic tests were negative except for the absence of Achilles tendon reflexes. Roentgenograms showed thinning of the right pedicle of the second lumbar vertebra and definite widening of the spinal canal at that level. Lumbar puncture yielded clear yellow cerebrospinal fluid containing 187 mg. of total protein per 100 ml. Arterial pulsations were reduced, the Queckenstedt test demonstrated a partial spinal subarachnoid block, and a cisternal myelogram showed a complete block at the upper level of the second lumbar vertebra. Lumbar laminectomy was performed and an ependymoma of the filum terminale was found and almost entirely removed. Postoperative roentgen therapy was given. Within a few months the patient returned to his trade as a barber, and when last seen, 10 years after the operation, he was working regularly, and his neurologic examination disclosed only slight residual sensory changes.

Case 2. A middle-aged man came to the Medical Division of the Cleveland Clinic in 1944; he gave a history of pain along the course of the left 12th thoracic nerve and in the back for almost a year. In order to obtain some rest at night, it was necessary for him to sit up all night and to take narcotics. Five inches of the left 12th rib and the 12th intercostal nerve had been removed without relief. The neurologic findings in 1944 were normal. The only abnormality of the spine evident on roentgenograms was arthritis. A diagnosis of myositis of the back muscles, and diagnoses of possible spinal arthritis, peripheral neuritis, and beriberi were made. A regimen to improve his general condition—vitamins, rest, dental care, and physical therapy—and Pantopon, gave only transient relief. Two years later he was examined in the Department of Neurological Surgery. He reported that the pain had persisted, and that he had received roentgen treatment to the spine without relief. The skin over the site of treatment was leathery and brown. There was no numbness or paralysis of the legs, and urination was not difficult. Flexion at the waist was limited. The patellar reflexes were sluggish, and the Achilles tendon reflexes were absent. Lumbar puncture yielded clear, colorless cerebrospinal fluid; there was an almost complete spinal subarachnoid block. The total protein content of the fluid was 85 mg. per 100 ml. Myelography using Pantopaque showed a partial block at the 9th thoracic vertebra, and laminectomy at that level revealed a cystic neurofibroma attached to a nerve root, with little evidence of compression of the cord. The tumor was removed. The patient returned to work a few months later, and continued to work without pain until his death from a "stroke" some years later.

Case 3. An 18-year-old girl experienced soreness and stiffness of the thighs, and pain in the coccyx and in the ankles for 14 months. Tonsillectomy had been done with the hope of eliminating a possible focus of infection. Taping of the back, rest on a hard bed for 2 weeks, a corset, section of the fascia lata in both hips, and an appendectomy failed to relieve the symptoms. Neurologic examination disclosed an absence of patellar and Achilles tendon reflexes; atrophy of both legs; a stiff, straight lumbar spine, and perverted sensation to touch in both feet. The patient was obviously in great pain. Lumbar puncture at the level of the 2nd and 3rd lumbar vertebrae caused pain, but no cerebrospinal fluid was obtained; at the level of the 1st lumbar vertebra, slightly bloody, yellow fluid was obtained, and there were normal pulsations and no subarachnoid block. The protein content of the fluid was 175 mg. per 100 ml. Myelography showed a complete block below the 1st lumbar vertebra, and lumbar laminectomy revealed thinning of the laminae and atrophic dura covering a large ependymoma of the filum terminale which filled the entire lumbar spinal canal. The tumor was partially removed, and a course of deep roentgen therapy was given after the operation. Twenty-two years later the patient was free from pain, but she had numbness of the left hip and the right foot. Her legs ached somewhat at night, but the ache did not interfere with sleep. Sphincter control was impaired. She was quite strong and was able to carry on her daily activities without difficulty.

Case 4. A well-built, strong man of 39 years was examined here because of pain in the upper left quadrant and stiffness and poor control of the legs for 5 years. There was no numbness of the legs or loss of sphincter control. Previous diagnoses had been "gas on stomach," ulcers, left inguinal hernia, and ruptured intervertebral disk. He had been advised to have an operation for hernia and also removal of a disk, but he refused these operations. For a year he had experienced increasing loss of control of the left hand when typing, and sharp, shock-like pains in the upper chest and shoulders. Neurologic examination disclosed slight spasticity of the legs, hyperactive patellar reflexes, an equivocal Babinski sign on both sides, slightly spastic gait, and no sensory loss. A diagnosis of pyramidal tract sclerosis was considered, but lumbar puncture yielded clear, straw-colored cerebrospinal fluid with a protein content of 170 mg. per 100 ml.,

and there was an almost complete spinal subarachnoid block. Myelography showed a complete block between the 7th cervical and 1st thoracic vertebrae and erosion of the left pedicles of these vertebrae. At laminectomy an "hour-glass" neurilemmoma of a cervical nerve was found to be compressing the spinal cord and extending laterally through an enlarged intervertebral foramen into soft parts of the neck. After removal of the benign tumor in 1948, the patient made a good recovery and returned each year for re-examination. When last seen, in 1959, he was in excellent condition, and there were no signs of recurrence of the tumor.

Case 5. A 42-year-old woman first noticed difficulty in walking 3 years before coming to the Cleveland Clinic. She was fitted with arch supports. Soon after, her legs began to ache. She consulted an orthopedic surgeon and was advised by him to have a neurologic examination; instead she tried salve for her feet, "nerve pills," and a trip to the country. She did not improve, and noticed the gradual development of numbness in the feet, the backs of the legs, and the saddle area. She also noticed an electric shock-like sensation in the lower back on jarring movements. She consulted a neurologist; he recommended a lumbar puncture, which she refused. Roentgenograms showed slight arthritis of the spine, and the patient took the baths at Magnetic Springs without relief. During the next 2 years she tried injections of vitamins, "spleen pills," and a trip to Florida. The numbness increased, and she consulted a physician who removed the coccyx, which was said to be fractured. Almost 3 years after the onset she consulted a neurologic surgeon, who thought that she probably had a ruptured intervertebral disk, and advised a lumbar puncture and an operation for the disk, both of which procedures were refused by the patient. She feared what she was pleased to call "experimenting." By this time she was thoroughly confused and unhappy and sought the advice of an osteopath who found one leg shorter than the other and a narrow space between the 4th and 5th lumbar vertebrae; he advised a lift for the left shoe and "radio treatments." Incontinence of urine and feces had started 2 or 3 months previously.

Neurologic examination at the Cleveland Clinic 3 years after the onset of her symptoms revealed hyperactive patellar reflexes, absent Achilles tendon reflexes, saddle anesthesia, and hypesthesia over the backs of both thighs and the lateral aspects of both feet, but no motor paralysis. Roentgenograms of the lumbar spine showed erosion of the lamina and of the posterior part of the body of the 1st lumbar vertebra. She was finally prevailed upon to accept lumbar puncture; there were no pulsations of the cerebrospinal fluid, a complete subarachnoid block, and a protein content of 95 mg. per 100 ml. Laminectomy of the 12th thoracic and first 2 lumbar vertebrae was carried out, and disclosed an extradural "hour-glass" neurofibroma compressing the spinal cord at the level of the 1st lumbar vertebra and extending through the intervertebral foramen beneath the paraspinal muscles into the retroperitoneal space behind the kidney. This tumor was removed, and the patient reported herself to be in good health 16 years after the operation.

Case 6. A 35-year-old man first noticed pain in the left hip and leg while turning a handspring 1½ years before examination here in 1931. At first the pain was intermittent, occurring chiefly at night, but later it was aggravated by coughing, sneezing, or straining at stool. It radiated into the left trochanter, anterior left thigh, and the anterolateral aspect of the left leg to the ankle. The pain became almost continuous day and night about 3 weeks before the patient was admitted to the Cleveland Clinic Hospital. Previous diagnoses included sacroiliac strain and sacroiliac arthritis. In 1931 the removal of "foci of infection" was in vogue, and all of the teeth had been extracted elsewhere without relief of his leg pain. "Electric" treatments were also unsuccessful. Neurologic examination disclosed no demonstrable sensory or motor changes in the legs, and the tendon reflexes were normal. Lumbar puncture yielded clear yellow cerebrospinal fluid, which did not pulsate, and there was a partial subarachnoid block. The fluid contained a marked excess of globulin, and a myelogram showed a definite delay in the descent of cisternally injected Lipiodol at the level of the 3rd lumbar vertebra. Lumbar laminectomy disclosed a neurofibroma attached to a nerve of the cauda equina, and it was removed. When last examined, 28 years later, the patient was quite well and free from pain.

SUMMARY

It has not been my aim here to present anything new or original in regard to the diagnosis or treatment of spinal cord tumors, many of which have been successfully removed since Victor Horsley first showed the way in 1887. This paper is merely a plea to all of us not to forget the possibility of such a lesion when we are attempting to analyze obscure and atypical forms of pain, with or without signs of neurologic deficit, and to bear in mind the extremely useful diagnostic function of the spinal puncture needle.

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Editorial

EARLY ATTACK ON SEVERE DISEASE?

Degenerative occlusive arterial disease is being attacked surgically and blood flow restored to ischemic areas with increasing success. Amongst the most spectacular, if uncommon, successes are those in which arterial blood flow is reestablished in occluded vessels to the gastrointestinal tract. There is ample evidence that operative removal of occluding material in the lumen of these arteries is feasible, but recognition of gradually increasing arterial insufficiency is difficult and forewarning of final complete block generally lacking. Patients frequently come to operation with large segments of intestine compromised, thrombosed vessels beyond the occlusion, and metabolic derangements accompanying peritonitis and malabsorption. If there were manifestations of relative insufficiency such as intermittent claudication in the lower extremities, angina in the myocardium or "little strokes" in the cerebrum, operation could be attempted earlier and under better circumstances.

Arterial flow to the intestine differs from that to the periphery. Portal venous blood may be delivered to the liver with a high oxygen content, sufficiently high to oxygenate the liver in the absence of hepatic artery flow. Extraction of oxygen in the course of flow through the splanchnic area is related to activity of the intestine. Presumably with total intestinal activity portal vein blood oxygen content would correspond to that in peripheral venous blood. This is a tremendous reserve afforded the intestine, a reserve which may account for partial occlusion of major vessels being inconspicuous. Furthermore, intercommunications between the branches of the main aortic trunks, coeliac, superior and inferior mesenteric arteries, are so efficient that gradual occlusion of 1 or 2 of the 3 creates little overt disturbance.

The main energy expending functions of the gastrointestinal tract dependent upon arterial blood are: secretion, muscular tonus, and peristalsis. Propulsion of chyle in lacteals is a secondary effect of the pumping action of peristalsis. It is difficult to determine from older people, who are likely to have occlusive abdominal artery disease, the subtle changes in dietary or eating habits which reflect interference with these functions. Accompanying cerebral vessel disease makes it impossible for them to remember what they ate 10 minutes before. A case could be constructed on a logical basis, however, for certain signs being attributable to arterial insufficiency.

It is accepted that as part of old age certain gastrointestinal difficulties arise. Constipation is common. Peristalsis most likely creates the greatest demand for arterial blood and could be the activity which corresponds to walking up a hill in lower extremity arterial insufficiency. Constipation which progresses to obstipation and obstruction is certainly a danger signal. Absorption of fat through lacteals is secondarily dependent on peristalsis. When fat is not absorbed in the gastrointestinal tract, the subcutaneous body stores are mobilized and burned, and thus the withered, thin, wrinkled skin appearance of old age is created.

When arterial insufficiency interferes with intestinal muscular tonus it well could be the reason for unexplained bouts of distension, bloating, and such consequences as sigmoid volvulus. Just as in the periphery, rest and cessation of demands for other purposes will restore a modicum of blood supply which promotes return of tone.

The most subtle change which may be of significance is loss of appetite or the development of peculiar eating foibles, both possibly associated with inadequate secretions. Old people will frequently choose to eat small quantities of carbohydrate, candy for instance, at frequent intervals and stubbornly refuse carefully prepared balanced meals. This may be all they can do with diminished circulation to their gastrointestinal tract and the way they stay alive. Carbohydrates are most easily absorbed in the upper intestine and with least peristaltic and secretory activity. The aggressive dietician forcing a balanced meal on such an oldster may precipitate necrosis of the bowel.

It would not be practicable to accept such intangible evidences of arterial insufficiency as

indication for a formidable operation on intestinal arteries. It is entirely logical, however, to consider under such conditions further investigation by aortography. Since this diagnostic measure is being developed and safeguards have been established, aortograms under these conditions can precisely pinpoint an area of occlusion at the aortic origin of one of the intestinal trunks, and operative endarterectomy can restore blood flow adequate for nutritional competence.

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Book Review

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Surgery in World War II, Neurosurgery, Volume II. By R. GLEN SPURLING, M.D., AND BARNES WOODHALL, M.D. Office of the Surgeon General, Department of the Army, Washington, D. C., 1959.

This is the second volume of the history of neurosurgery in World War II. Volume I, published in 1958, is concerned with the administrative details of military medicine; the evolution of neurosurgical policies, and head trauma, including certain of its immediate sequelae. This second, and final, volume is concerned with injuries and diseases of the spinal column and peripheral nerves. There is also a chapter on lesions of the intervertebral disk.

In the medical history of World War I, management of spinal cord injuries was one of the more discouraging areas. In one series of 32 cases reported by Colonel Harvey Cushing there was a 71.8 per cent mortality and fully 80 per cent died within the first few weeks as a result of operative mortality, decubitus ulcers, and urinary tract infections. As a direct contrast to this in World War II, of 482 patients with spinal cord injuries who reached field and evacuation hospitals of the First United States Army alive, there was an overall mortality of 14.5 per cent. Similar low mortality percentages were recorded in other evacuation hospitals. The reasons for this marked, and heartening, decrease in mortality are well described in chapters on general care of patients with spinal cord injuries and associated paraplegic problems, especially decubitus ulcers and urologic infections.

Part II of this volume contains probably the most complete and detailed account of peripheral nerve injuries and repairs available today in the literature. The injuries have been broken down as to nerves involved, type of treatment, time lapse before treatment, and results of treatment. When this data is analized in association with the Peripheral Nerve Registry (appendices E and F of this volume), full realization of the magnitude of this project in patient care can be obtained. The total number of nerves on which reports have been analized is 7,050. Almost one-third of these involved the ulnar nerve. Median (19.5 per cent), sciatic (16.9 per cent), and radial (14.1 per cent) were the next most common in frequency. In approximately 99 per cent of the cases, end-to-end anastomosis was performed as the initial treatment. Primary nerve suture was not attempted, but whenever possible, repair was performed overseas, after debridement and soft tissue wound healing. It is shown that the place of repair is not important, but that timing of repair is. Nerves repaired within the optimal time period of 21 to 90 days after wounding were restored to functional usefulness in countless cases in which the plan of delayed surgery usually practiced in World War I would have left useless extremities.

Neurosurgery furnished an especially brilliant chapter of military medicine in World War II and these two volumes document that history in a thorough and complete manner.

CHARLES M. HENDERSON, M.D.

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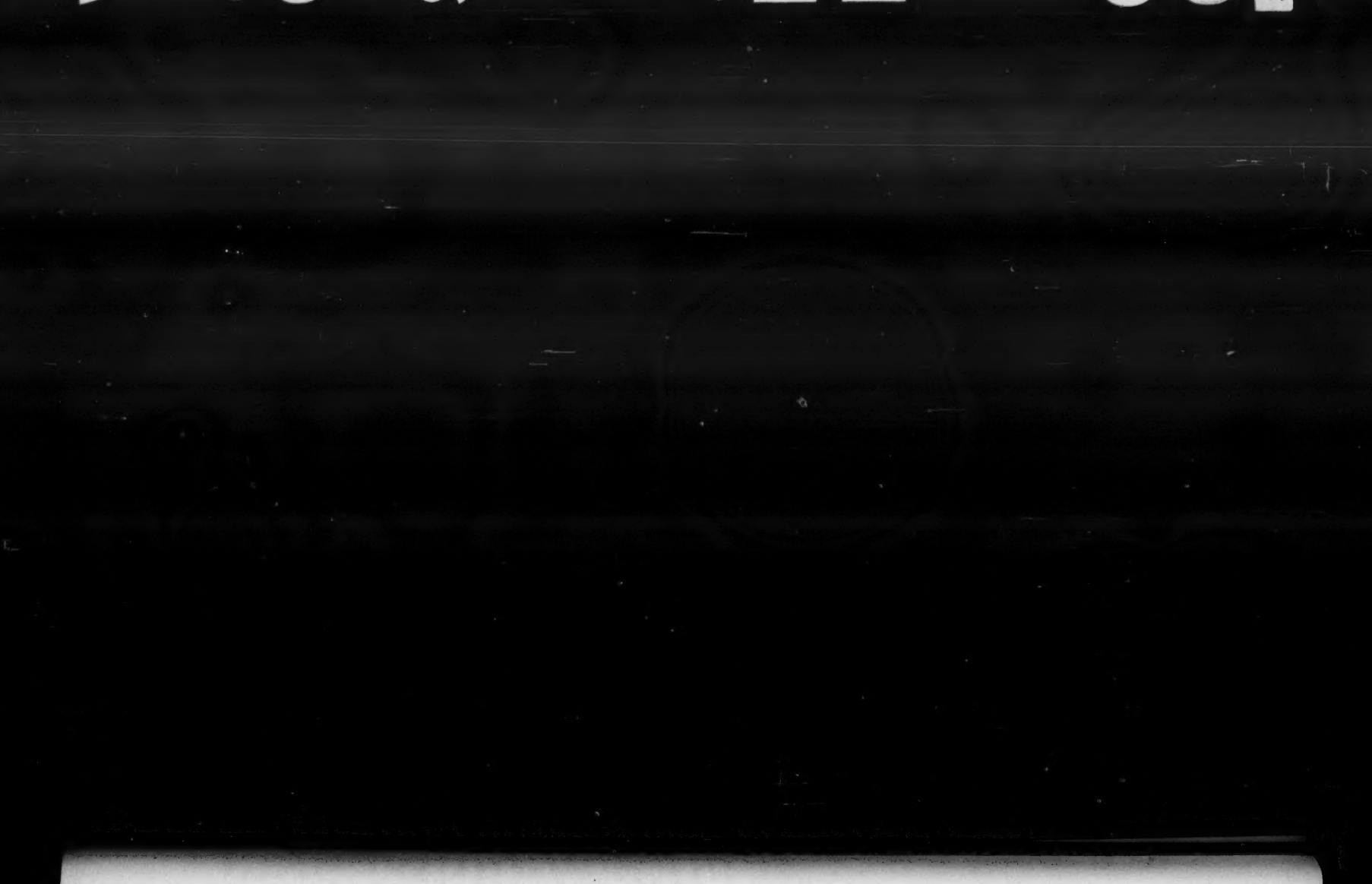
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